Vol. LXXXVII JUNE, 1928 No. 6

JUN 16 1928

ANNALS of SURGERY

A Monthly Review of Surgical Science and Practice

Edited by
LEWIS STEPHEN PILCHER, M.D., LL.D.
of New York
With the Association of
JAMES TAFT PILCHER, B.A., M.D.
and the Collaboration of
W. SAMPSON HANDLEY, M.S., M.D., F.R.C.S.
of London

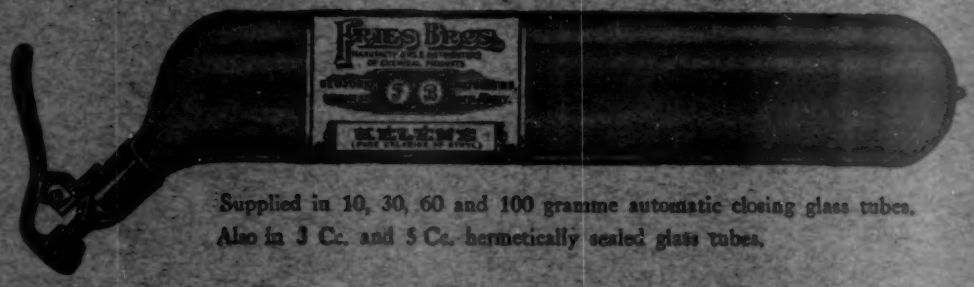
EPINEPHRECTOMY (ADRENALECTOMY) FOR HYPERADRENALINEMIA . . . 801
Prof. Vladimir A. Oppel . . . Leningrad, Russia
BLOOD FROM TRAUMATIZED LIMBS . . . 806
Dallas B. Phemister, M.D. . . . Chicago, Ill.
SPINA BIFIDA AND CRANIAL MENINGOCELE . . . 811
Thomas A. Shallow, M.D. . . . Philadelphia, Pa.
HEART INJURIES—WITH SUTURE . . . 823
Herbert H. Schoenfeld, M.D. . . . Washington, D. C.
PRIMARY HYPERNEPHROMA OF THE LIVER . . . 829
Irvin Abell, M.D. . . . Louisville, Ky.
REMOTE RESULTS OF BILIARY SURGERY . . . 837
F. Gregory Connell, M.D. . . . Oshkosh, Wis.
NON-PARASITIC CYSTS OF THE LIVER . . . 844
Alexius McGlannan, M.D. . . . Baltimore, Md.
PHLEGMONOUS GASTRITIS . . . 848
John J. Morton, M.D. and S. J. Stabins, M.D. . . . Rochester, N. Y.
LATE RESULTS IN PERFORATED AND GASTRO-DUODENAL ULCERS . . . 855
Richard Lewisohn, M.D. . . . New York, N. Y.
ASEPTIC END-TO-END INTESTINAL ANASTOMOSIS . . . 861
James K. Shen, M.D. . . . Peking, China
ACUTE INFLAMMATORY OBSTRUCTION OF TERMINAL ILEUM . . . 867
Clyde A. Roeder, M.D. . . . Omaha, Neb.
FIBROMATA OF THE MESENTERY . . . 870
William Edgar Darnall, M.D. . . . Atlantic City, N. J.
LYMPHANGIOMA OF OMENTUM . . . 872
William H. Fisher, M.D. . . . Toledo, Ohio
LYMPHOSARCOMA OF THE MESENTERY . . . 879
L. L. Bigelow, M.D., E. Scott, M.D. and S. W. Obenour . . . Columbus, Ohio
BRACHIAL PLEXUS ANÆSTHESIA . . . 883
Prof. D. Kulenkampff, Zwickau, Germany and M. A. Persky, M.D. . . . Providence, R. I.
TRAUMATIC OSTEOPOROSIS OF THE CARPAL BONES . . . 892
Joseph Buchman, M.D. . . . Brooklyn, N. Y.
DERANGEMENTS OF THE KNEE-JOINT . . . 911
Melvin S. Henderson, M.D. . . . Rochester, Minn.
MELANOTIC TUMORS . . . 917
Alec Horwitz, M.D. . . . Rochester, Minn.
MALIGNANT LYMPHOCYTOMA . . . 934
Philip G. Silver, M.D. . . . Montreal, Canada
RECURRENT FIBROMATOUS TUMORS OF THE SKIN . . . 945
David A. Willis, M.D. . . . Chicago, Ill.
TRANSACTIONS OF THE NEW YORK SURGICAL SOCIETY . . . 950
Stated Meeting Held February 22, 1928
BRIEF COMMUNICATION: *Rogoff*: Critique on Adrenalectomy for Alleged Hyperadrenalinemia . . . 959
INDEX . . . 961

J. B. LIPPINCOTT COMPANY, PUBLISHERS

KELENE

(Pure Chloride of Ethyl)

For Local and General
ANESTHESIA



Supplied in 10, 30, 60 and 100 gramme automatic closing glass tubes.
Also in 3 Cc. and 5 Cc. hermetically sealed glass tubes.

The automatic closing tubes require no valve
Simply press the lever

Widely used for many years

Sole Distributors for the U. S. A. and Canada
MERCK & CO. INC.
Philadelphia New York St. Louis Montreal
Main Office: Rahway, N. J.

Wherever Indicated

Gray's Glycerine Tonic Comp.

(Formula Dr. John P. Gray)

—Restores depleted energies
—Correlates functional activity
—Stimulates metabolism

The Purdue Frederick Company, 135 Christopher St., New York

ANNALS of SURGERY

VOL. LXXXVII JUNE, 1928 No. 6

EPINEPHRECTOMY (ADRENALECTOMY) FOR HYPERADRENALINEMIA IN SPONTANEOUS GANGRENE*

BY PROF. VLADIMIR A. OPPEL

OF LENINGRAD, RUSSIA

FROM THE SURGICAL CLINIC OF THE MEDICAL ACADEMY OF LENINGRAD, U.S.S.R.

FOR a long time the origin of the so-called cases of spontaneous gangrene has remained in obscurity. It has always been attended with serious consequences and usually ended in the amputation of the affected extremity if, as so often happened in the meantime, the patient did not die from intercurrent infection either from the effect of the toxins on the circulatory system or from gangrene of the intestines or thrombosis of other arteries.

It is essential to appreciate that in the beginning, spontaneous gangrene invariably is evidenced as a dry process and seldom becomes moist. This differentiation is necessary to remember as it will be noticed that Buerger's disease, termed "thrombo-angiitis," appears to be an appreciably different process from a spontaneous gangrene. Usually the thrombi develop in the superficial veins of the extremities, although the condition Buerger describes, as a rule, goes through a process of dry gangrene, the reason in both instances being similar and apparently due to the fact that there is an interference of the afflux of arterial blood to the peripheral extremities caused by thrombosis of the arteries. Occasionally the thrombosis of the superficial veins can be noticed in some of the patients but it is unusual. The thrombosis of the deep veins is rarely observed, while in cases of spontaneous gangrene the additional thrombosis of the deep veins changes it from the dry form into a wet one.

In cases of spontaneous gangrene in which I place those cases of thrombosis of the deep arteries, in order to increase the blood in the peripheral extremity, for a long time I have practiced the bandaging of the veins in the region of the hip. This has been likewise done by various other surgeons on many other occasions and it has been noticed that it usually gave some temporary benefit in that there showed a definite hyperæmia particularly noted on the dorsum of the foot and a general suffusion in the extremity with a consequent amelioration or disappearance of pain. Based on the above effect of bandages applied about the knee or hip, a series of palliative operations have been evolved such as the periarterial sympathectomy of Leriche and the severance of the sensory nerve trunks as developed by Molotkoff.

In instances of spontaneous gangrene, the arteries are similarly narrowed

* Translated by Dr. Alexander S. Kasatchenko of Montreal, Canada.

or in a state of spasm and our efforts should be directed to any method which will cause dilatation of arterial collaterals. Previously, the surgery of many conditions of this type was developed on the pathological anatomy noted in the affected arteries and it had been appreciated frequently that in these cases the femoral artery, for instance, seemed to be especially small. It was therefore felt that the condition was the result of a congenital narrowing of the arterial lumen. This theory has, of course, now been rejected. Appreciating that in cases of spontaneous gangrene, the immediate cause was due to a blocking of the artery, caused the German surgeons to institute studies against what they termed "endarteritis obliterans" in which they demonstrated that the obstruction was caused by thrombi which extended both in an upward and a downward direction. This hypothesis of the development of this type of gangrene has been generally accepted, that thrombosis affecting first the arteries of the extremity and then continuing in other parts of the body so that it may develop further in the heart, intestines, brain and kidneys, etc. The question, however, arose, "What was the original etiology of the primary thrombus?" Buerger was inclined to attribute it to racial idiosyncrasy as most of his cases occurred in Jewish immigrants. However, the condition has been found in many other races and is particularly prevalent in the northwestern part of the United States of Soviet Russia. It would, therefore, be apparently more reasonable to consider spontaneous gangrene as rather one of geographic than racial distribution. Irrespective of this factor, Buerger failed to explain the reason for the thrombus and what caused the disease of the arterial wall which, in some cases, terminated in a sclerosis of it, as was demonstrated definitely by T. von Manteufel, although in 1911 I advanced an hypothesis relative to the causation of this condition by considering it an hyperfunction of the adrenal bodies and that essentially the condition was caused by an hyperadrenalinemia. This was, however, met with severe criticism and I have attempted to justify my position by evolving proof both clinically and experimentally.

I termed the disease of the artery an arteriosis and hypothecated that under the influence of the stimulation of an excessive amount of adrenalin in the blood, the arteries were in a state of spasm which caused a derangement of the degree of nourishment to the arterial walls, with a consequent erosion of the endothelium and a resulting focus of lowered resistance of the muscularis with the eventual establishment of the so-called arteriosis, the succeeding formation of sclerosis, and the development of the local thrombus which, continuing to increase in size, eventually impinged upon the lumen of the artery, growing in both directions. Before starting to develop proof of the etiology of the condition being due to an hyperadrenalinemia, I wish to compare symptomatically spontaneous gangrene and Buerger's disease taking chiefly into consideration an article published by Vaquez who described the symptom complex of Buerger's disease in great detail and who was one of my harshest critics.

In Buerger's disease, there occurs an hyperglobinemia. This has similarly

been noticed by Gleberich in cases of spontaneous gangrene, or as I have termed it, hyperadrenal arteriosis in which condition there is very characteristically an hyperthrombositosis which has been calculated as high as one half million thrombocytes. It would be interesting to know to what extent this holds good in instances of Buerger's disease. In both conditions, there is apparently an hyperglycemia present as demonstrated by Obraszoff. In both conditions there is an increased viscosity of the blood which was demonstrated by Ditrogorsky. There is, in at least one-half of the cases of hyperadrenal arteriosis, a definite inhibition of the coagulating time. Parenthetically, I would like to call attention to the fact that hyperthrombositosis facilitates the formation of white thrombi and also that increased viscosity and accelerated coagulation time facilitate stratification of red thrombi. In Buerger's disease there is an increase of cholesterin in the blood. This was not noted in instances of hyperadrenal arteriosis (Sokoloff). It does not seem to me that this difference should be considered a valid reason for distinction between the two conditions when in the remainder, the findings appear identical. In addition, in typical instances of hyperadrenal arteriosis, there is an increased central arterial pressure as can be determined on the shoulder while there is a lowered peripheral pressure found in the fingers, which can be explained if the arteries are partially passable or if there is only an incomplete spasm of the peripheral arteries, also that in these cases the central arterial level of the blood is ordinarily higher and reaches 12 and even 13 milligrams of one per cent. After following Collip's investigation, it would seem feasible that the increased level of the central arterial blood tends to contribute toward a falling back of it to the side of arteries affected by hyperadrenalinemia. Is it not possible further that in such cases where we find an hyperfunctioning of the adrenals, might there not similarly be an hyperfunctioning of the epithelial bodies in general? It therefore seems to me that there is no real difference between Buerger's disease and hyperadrenal arteriosis, that they are both the same condition which can best be given the nomenclature of spontaneous gangrene.

Feeling that the above hypotheses and deductions were correct, I have for some time, for the cure of this disease, been performing a left-sided epinephrectomy (adrenalectomy). I have performed this operation on one hundred and thirty cases. At the present time there has been thus far but a very small mortality. In the last seventy epinephrectomies performed on account of hyperadrenal arteriosis or spontaneous gangrene, only two patients have died.

It has been noted that after epinephrectomy, the number of thrombocytes decreases, the level of the blood sugar decreases, even when it is normal at the time of operation, a reduction having been noted in one instance from .095 per cent. to .08 per cent. Co-incidentally, the central arterial pressure becomes lower but the peripheral blood pressure on the contrary rises, thus diminishing the balance because it would appear that as the result of the

peripheral arteries being dilated, the wave of blood attaining the periphery meets with less resistance.

When we realize that all the above enumerated data are to be observed during the course of the first weeks after epinephrectomy, which is time enough for all influences of an operation on the body to cease, it seems to me that it is fair to assume that the hyperthrombocytosis, hyperglycemia, increased central arterial pressure and lowered peripheral pressure were all dependent upon the hyperfunctioning of the adrenal gland. Further, it has been repeatedly noted that a pulse which before operation could not be palpated, has post-operatively re-appeared in the arteries, which is additional proof that they were in a state of spasm. The real proof of my contention is indeed to be noted when one sees a patient with definite gangrene of the fingers or foot and in excruciating pain in whom immediately after epinephrectomy, the gangrene stops advancing, the extremity becomes pink, the dead tissues slough away, the pain stops and he is able to get around with an extremity which had previously been condemned to amputation.

It appears to me in stating the above clinical facts that my contention has been sufficiently justified and that the correctness of my hypothesis has been proven as far as is possible, that the basis of these instances of spontaneous gangrene or Buerger's disease is beyond any reasonable doubt, due to an hyperadrenalinemia. The actual proof, of course, can only be ascertained by the isolation, detection and quantitative estimation of the amount of adrenalin in the blood.

It is a most difficult task to determine the presence of the hormones in the human blood of any one suffering from an hyperfunction of any of the glands of internal secretion. The method employed by my assistants, Doctor Achutin and Doctor Ornatsky, in overcoming this difficulty, is as follows: the blood to be examined is used on an isolated (method of Magnus) knot of gut of a cat, upon which the blood of any healthy animal such as a rabbit, or of a normal man, causes a convulsive abridgment (curtailment) followed by paralysis, while the blood of a patient suffering from hyperadrenal arteriosis or the blood of a patient suffering from Raynaud's disease or the blood of a rabbit artificially adrenalized, provokes a spasm in the gut and does not paralyze it. In other words, the gut continues the abridgment (curtailment) even sometimes in the presence of an increased tonus. It is interesting to note that a similar phenomena is provoked with the blood of a man in good health into whom adrenalin has been injected and vice versa, the blood of an epinephrectomized patient gives the same reaction as that of a man in good health. It is to be hoped that a more definite quantitative method for analysis may be evolved eventually so that even more definite proof can be given of my theory.

Assuming, therefore, that I am correct in my inferences and deductions as based upon the above experimental and practical results, it seems proper to place both hyperadrenal arteriosis and Raynaud's disease in the same category. There are two substantial distinctions, however, to be made between the two

conditions in that the hyperadrenal arteriosis occurs chiefly in the male while Raynaud's disease is more ordinarily noted in the female. The second is that in the arteries in hyperadrenal arteriosis thrombi are formed while in Raynaud's disease they do not become thrombosed. Furthermore, in the former condition, the arteries of the lower extremities are first affected but in Raynaud's disease in many instances the upper extremities are involved, although in both conditions the general arterial system is affected. It is certainly difficult to explain why this condition reacts differently on the different sexes and may possibly find its explanation in the influence of the sex hormones. The fact, however, I feel sure remains that both diseases are due to an hyperadrenalinemia.

For the relief of Raynaud's disease, I am also employing epinephrectomy with satisfactory results, in that where before operation the extremities were cold and bluish-black, within one or two days post-operative they became warm and pinkish. The pains in the extremities disappear and the patients are able to resume their occupations. I recall that Doctor Brown of the Mayo Clinic has obtained satisfactory results in these cases by lumbar sympathectomy and in Russia several surgeons like Shamoff and Abrajenoff have obtained satisfactory results after Leriche's operation. Indeed, Leriche, in his last publication, states that epinephrectomy has given results in these cases where his operation of peripheral arterial sympathectomy has been ineffectual. I have performed personally, at least ten lumbar sympathectomies and can confirm Doctor Brown's results that the temperature of the extremities increases and that the condition is certainly improved. This is quite conceivable in that by this operation the sympathetic system is interrupted, removing the inhibiting influences on the arterial musculature and allowing them to dilate. In, however, performing epinephrectomy, I endeavor to accomplish the same result, but for the entire body and for all extremities as well as for the internal organs and it has been noted very strikingly that in patients with Raynaud's disease there is a definite leveling of the functions of the heart, it being reduced in size where previously it was dilated and the entire tone becoming more distinctive, similar observations having been noted in instances of hyperadrenal arteriosis.

It is apparent that as the result of these observations the *modus operandi* of the condition of spontaneous gangrene would seem best accounted for by the hypothesis of hyperadrenalinemia irritating the sympathetic nervous system with resultant spasm of the arteries and the consequent pathology.

It is to be hoped that my view may be confirmed by American surgeons and that they will employ in instances of spontaneous gangrene the uncomplicated and apparently efficient operation of epinephrectomy.

NOTE: The reader is referred to page 959, for a communication from Dr. J. M. Rogoff of the Experimental Laboratory of Medicine, Western Reserve University, Cleveland, Ohio, entitled "A Critique on Estimation of Adrenalin in the Blood," for a valuable sidelight on this important subject.

EDITOR.

THE VASCULAR PROPERTIES OF TRAUMATIZED AND LAKED BLOODS AND OF BLOOD FROM TRAUMATIZED LIMBS*

By Dallas B. Phemister, M.D.

of Chicago, Ill.

If the circulation to a limb is obstructed its arterioles and capillaries dilate. On release of the obstruction, reactive hyperæmia follows, lasting about as long as the obstruction, during which time the limb is flushed and increased in volume and the arterial limb pressure is lowered. It occurs equally well whether the limb is innervated or denervated. What causes this vasodilatation? Anrep, Lewis and Grant have assumed that a vasodilator metabolite—histamine or a histamine-like substance—is formed in the asphyxiated tissues during the period of hyperæmia. This substance is either destroyed *in situ* or washed away in the venous blood. To test such blood for vasodilator action, use was made of Anrep's viviperfusion apparatus. This consists of a flask attached to a cannular system which can be inserted in the course of an artery. The flask can be shortcircuited by means of a T bone-tap, emptied and filled, and its contents then circulated by again adjusting the tap. The apparatus was inserted in the course of the femoral artery of a dog and permitted of recirculation of blood collected from the femoral vein during the period of hyperæmia after recovery of the limb from the effects of the obstruction. The dog was anæsthetized with ether and the limb was denervated to exclude nervous influences, so that the changes observed must be explained on a physical or chemical basis; 0.4 of a gram of heparin was injected to prevent coagulation of blood and the perfusion apparatus was inserted in the course of the femoral artery just below Poupart's ligament. For the collection of venous blood either a Y cannula or a second perfusion apparatus with flask was inserted in the course of the femoral vein. A plethysmograph was applied to the limb for measuring limb volume, a mercury manometer was connected with the cannula in the femoral artery and another with a cannula in the carotid artery. Limb volume, limb pressure and carotid pressure were recorded on a long paper kymograph. The results of certain experiments with this method were reported by Phemister and Handy (*The Journal of Physiology,* 1927, vol. lxiv, p. 155). It was found that the circulation of the nontraumatized (unshaken) blood collected from the femoral vein of the limb during the period of hyperæmia caused no vasodilatation; that is, if a vasodilator substance is formed in the tissues during the period of obstruction it is not washed out by the blood to an extent which makes its presence demonstrable by this method. Ordinary venous blood behaves likewise.

* Read before the American Surgical Association, May 14, 1927.

If either arterial or venous blood flowing through the flask is shortcircuited for a period up to four or five minutes and the stagnant blood then sent through the limb (in case of venous blood the flask with its tubes clamped off must be transferred to the arterial side), it causes either no vascular changes or the changes are very slight. If either arterial or venous blood is withdrawn and shaken for a few seconds to a few minutes before insertion into the flask, it produces vasodilatation on circulation. If either arterial or venous blood is shortcircuited in a flask containing a bead, the inlet and outlet tubes clamped and the flask detached and shaken without exposure to either air or light, it produces vasodilatation. Arterial or venous blood, the gaseous content of which has been modified by exhaustion of gases and recharging with varying quantities of O_2 or CO_2, always caused vasodilatation. However, the blood was always traumatized in making the modification.

The longer the traumatism the greater the vasodilatation up to a certain point. The maximum effect from shaking is reached in two to three minutes, beyond which shaking for as long as ten minutes produces no increase.

What change comes about in the blood as a result of this amount of traumatism that gives it this vasodilator property? Have vasoconstrictor substances been destroyed, or has a vasodilator substance been liberated or created from blood elements? The two vasoconstrictor hormones, adrenalin and pituitrin, are far too stable from chemical standpoints to be destroyed by such slight traumatism as is necessary to give the reaction, and when blood has been traumatized to a point where it gives a maximum dilator action it is necessary to add at least five times as much adrenalin or pituitrin as is necessary to give vasoconstriction in order to obliterate this vasodilator effect.

Is a vasodilator substance liberated or created by traumatizing the blood elements? When blood is shaken for two to three minutes which is enough to produce a maximum vasodilator effect, the pH remains constant or practically so; consequently, the reaction is not the result of change in the acid-base balance.

Is histamine or a histamine-like vasodilator substance liberated from the blood cells by breaking them up? If so, the greater the amount of traumatism the greater the amount of such substance liberated and the greater should be the vasodilator effect. Laking, or hæmolysis of blood by freezing and thawing, breaks down nearly all of the cells and should most effectively liberate the vasodilator substance. Also, the greater the amount of traumatized blood circulated the greater should be the vasodilator effect. Consequently, quantitative tests were made by the circulation of different amounts of blood traumatized for varying lengths of time, of laked blood and of blood to which different amounts of histamine were added. When more than 75 c.c. were used, blood was added from another dog to keep blood volume up to normal. It was found that the greater the amount of histamine added to a given quantity of blood and the greater the quantity of histaminized blood circulated the more marked the vasodilator effect. With large amounts of histaminized blood entering the animal there was an initial increase in limb volume, fall

in limb pressure and then fall in general blood-pressure. But after one to one and a half minutes both limb and general vascular beds would acquire the power of resisting much of the histamine action, although when 500 c.c. of blood were used the histamine would continue to enter the circulation in much the same concentration for three and a half to four minutes.

The quantitative tests with traumatized blood were made as follows: Blood was traumatized by shaking for a few seconds to one hour and the effects were determined of circulating amounts varying from 30 to 475 c.c., as previously stated. When 30 c.c. were used the maximum vasodilatation was produced by shaking for two to three minutes; prolongation of the act for ten or twelve minutes producing no alteration in the effect; but shaking for twenty minutes caused vasodilatation followed by vasoconstriction, and shaking for forty to sixty minutes caused vasoconstriction from the beginning, which lasted for three to five minutes, after which vascular response was very much diminished.

Increase in amount of slightly traumatized blood circulated did not increase proportionately the vasodilator effect. Thus 30 c.c. of blood shaken for two or three minutes cause vasodilatation, which reached its maximum in twenty to twenty-five seconds and disappeared in fifty to seventy seconds; 75 c.c. produced slightly more effect than did 30 c.c., but 220 or 475 c.c. produced no greater effect than did 75 c.c.; 475 c.c. of blood traumatized for two to five minutes caused a maximum dilatation in thirty to thirty-five seconds, which wore off in sixty to ninety seconds despite the fact that undiluted traumatized blood was still going through the limb; however, when 475 c.c. of blood shaken in 50-c.c. lots for one hour were circulated there was limb constriction lasting for eight minutes followed by slow recovery.

Thus blood that had been slightly traumatized caused vasodilatation lasting a short time, and increase in the quantity of blood circulated beyond approximately 75 c.c. did not increase the duration of the vasodilatation; blood that had been traumatized extensively, as by shaking for twenty minutes, caused vasodilatation followed by vasoconstriction, and blood traumatized very severely, as by shaking for one hour, caused vasoconstriction only. The greater the quantity of severely traumatized blood circulated, the longer the duration of the vasoconstriction.

Quantitative tests were made with laked blood, which was prepared by freezing with liquid oxygen and thawing. Amounts were circulated varying from 1 minim to 220 c.c. One to 10 minims injected into the tube caused vasodilatation, which increased with increase in the amount of blood injected; but 1 c.c. caused beginning limb dilatation followed by limb constriction and 10 c.c. or more caused limb constriction only, and the greater the amount the longer the constriction lasted.

When untraumatized blood is treated with very small amounts of laked blood it produces vasodilatation on circulation, but as much as 5 to 10 per cent. produces beginning vasodilatation, followed by limb constriction and larger percentages cause limb constriction only.

Since blood traumatized in varying degrees acted similarly to blood that had been laked in varying degrees, it was thought probable that their action was produced by the same thing and that traumatism had produced hæmolysis. Consequently, blood that had been shaken for lengths of time varying from a few seconds to one hour was centrifuged and the plasma examined for the presence of hæmoglobin. It was found to be present in amounts that varied directly with the amount of traumatism, ranging roughly from 0.0573 gm. per 100 c.c. for blood shaken for thirty seconds to 3.689 gm. per 100 c.c., or over 20 per cent. of the total hæmoglobin for blood shaken for fifty-five minutes. Microscopic examination also showed evidences of breaking down of red cells. Consequently, the hæmolysis is responsible for the vasomotor properties of both traumatized and laked bloods.

The facts that small amounts of hæmolyzed blood or of traumatized blood caused vasodilatation and fall in regional blood-pressure when circulated through the limb raised the question as to their effect when injected into the general circulation. To determine this point, 475 c.c. of blood shaken for four or five minutes, or to which 5 to 10 c.c. of hæmolyzed blood were added, were circulated from a flask connected with the femoral vein. In none of several experiments was there any considerable amount of alteration in general arterial blood-pressure. It was thought possible that the immediate reactions from indirect blood transfusion in man might be due to the traumatism resulting from mixing with the sodium citrate solution; but the absence of any considerable fall in general blood-pressure when traumatized blood was inserted into the general circulation in dogs argues against that view.

Traumatic shock has been regarded by many investigators as due to a toxic substance derived from the traumatized tissues entering the general circulation and producing vasodilatation with resultant low blood-pressure. It might even be conjectured that a vasodilator substance is absorbed from the damaged extravasated blood in quantities sufficient to produce a state of shock.

A test for the presence of vasodilator substances in the blood from a traumatized limb was made in the following way: A viviperfusion apparatus was inserted into the femoral vein of a dog, a constrictor was applied distally and the limb was severely hammered. A second animal was prepared in the usual way with a viviperfusion apparatus in the femoral artery and a plethysmograph on the limb. Carotid blood-pressure tracing was then established for the first dog and the constrictor removed. The limb swelled rapidly afterward and the animal's blood-pressure gradually fell in the course of a few minutes to one hour to the level of 80 to 50 mm. of Hg., which is within the range of that ordinarily encountered in traumatic shock. The venous blood flowing through the flask was then shortcircuited, the connecting tubes clamped, the flask detached and connected with the artery of the second dog without traumatizing blood in the transfer. Circulation of such blood several times in three experiments usually caused very little or no vasodilatation; but if the blood was withdrawn from the flask and shaken for a short time it produced the usual vasodilator reaction. Therefore, if there is a vasodilator

substance in the blood coming from the traumatized limb of an animal which produces shock, it is not present in sufficient quantity to be detected by this method.

The experiment does not support the theory that shock is a traumatic toxemia from a histamine-like substance formed in the traumatized tissues and causing vasodilatation of the general capillary bed. Furthermore, necropsy examination of the traumatized limb showed increase in limb volume from hemorrhage which was sufficient to account for the fall in blood-pressure. In fact, the volume of blood that it was necessary to withdraw intermittently in the course of an hour in order to kill an animal was always less than the increase in volume of the traumatized limb of the other animal, which was due very largely to hemorrhage in the tissues.

The nature of the substance or substances liberated by traumatizing or laking the blood which produce the vascular changes here recorded have not been determined, but they appear to be the same in both cases. They are rendered active by the hæmolysis. The vasodilatation which results from a small amount of hæmolysis may be due to one factor, and the vasoconstriction which results from a large amount of hæmolysis may be due to another factor, or they may both be due to the same thing acting in different concentrations. The quantitative tests speak against the action being the result of histamine or a histamine-like substance.

No definite clinical phenomena can at present be explained by these reactions. But since such marked vasodilator properties can be created in the blood by mild injury and slight hæmolysis, it is not unreasonable to assume that these properties may be acquired in certain cases of injury or disease and that they may be responsible for certain unexplained circulatory symptoms.

SPINA BIFIDA AND CRANIAL MENINGOCELE *

By Thomas A. Shallow, M.D.
of Philadelphia, Pa.

In presenting the subject of spina bifida and cranial cephalocele, a topic which is so intimately associated with the names of Von Bergman, Von Recklinghausen, Hildebrand and Frazier, one must hesitate before he attempts to add to the extensive study and the work done by these men.

We are presenting these cases out of a series of sixty patients which have been observed in the J. Chalmers DaCosta Clinic of the Jefferson Hospital in which the question of operation arose.

Spina bifida is a congenital malformation of the vertebral column. It is

Fig. 1.—Spontaneous rupture of meningomyelocele.

usually located in the midline posteriorly, rarely the defect is found through the body of the vertebræ anteriorly. It varies from an arrested development of the arches of the vertebra to a complete absence of these structures. It is associated with a protrusion (known as a sac), usually in the midline and distended by cerebrospinal fluid. The contents of a sac may be cerebrospinal fluid alone, cerebrospinal fluid and cord or the cauda equina.

There are four common types of spina bifida. (1) Spinal meningocele, (2) meningomyelocele, (3) syringomyelocele, (4) spina bifida occulta.

(1) In a spinal meningocele, the simplest of these four types, the skin is usually sound over the entire sac. The dura and arachnoid may form the second and first layers of the wall of the sac, or the arachnoid alone may be present in the wall, the dura being present only at the neck of the sac. In meningocele the cord and nerves remain in the spinal canal.

* Read before the Philadelphia Academy of Surgery, January 16, 1928.

(2) In a meningomyelocele, the type which we have most frequently encountered, the skin was not sound in any of our cases. The dural layer was observed in one of several positions: (a) The dura was a simple cleft, remaining in the spinal canal and the sac protruding through the cleft. (b) The dura was cleft and blended with the fascia of the erector spinæ mass muscles. (c) The dura extended upward as a cuff and surrounded the neck of the sack for about an inch. The spinal cord or the cauda equina was found adherent to the arachnoid and the cerebrospinal fluid was in front of the nerve structures which were spread out and intimately blended with the posterior wall of the sac. In some cases the sac contained, in addition to the cauda equina or cord, nerve roots which passed from the spinal cord into the sac and then reëntered the spinal canal. In this type of case we found that there was a partial arrest of development of the nervous elements of the cord.

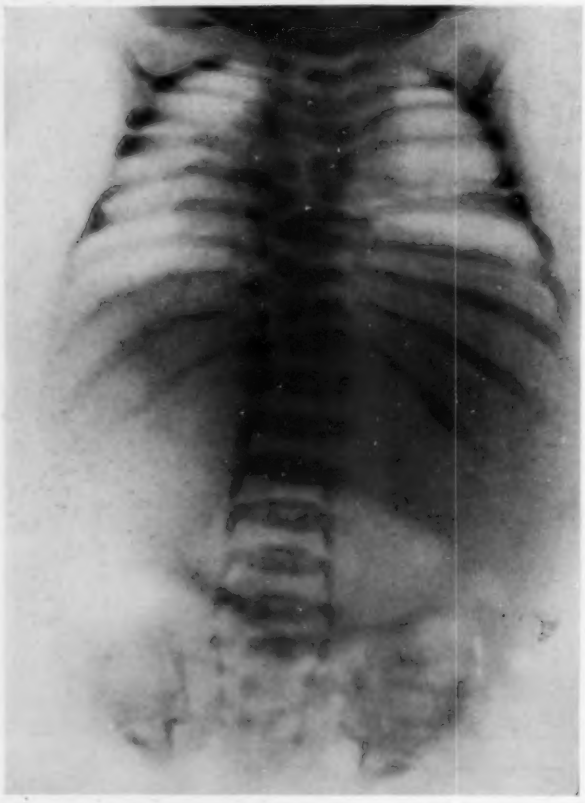


FIG. 2.—Showing defective development of 6–7–8–9–10 thoracic vertebræ—same patient as Fig. 1.

(3) Syringomyelocele. In this type we found that the central canal of the cord contained cerebrospinal fluid. The rest of the cord was spread out over the distended central canal. Both of these structures, cord and distended canal, passed through a dural defect.

(4) In the type known as spina bifida occulta, there is a spinal defect, discovered by touch and X-rays, which is covered with sound skin. Sometimes the spinal defect is marked by a depression of the skin. The cleft, in the occult form, is occupied by blended membranes adherent to skin and cord.† There is often a superabundance of hair over the lesion and tumor formation is common.

The etiology of such anomalies is failure of mesenchymal fusion. From the mesenchyme are derived the muscles of the back, the bones and ligaments

† Spencer and Gask, 1910, Blakiston & Son.

of the vertebral column and the meninges. That developmental arrest is the only cause of these anomalies is doubtful. Spencer and Frazier both emphasize the possibility of an excess of cerebrospinal fluid being a factor. In confirmation of this view we have seen spina bifida associated with hydrocephalus and hydrocephalus follow operations for spina bifida, and we have in our series observed hydrocephalus follow a spontaneous rupture of the sac. (Case II.)

FIG. 3.—Wide separation of cranial bones after spontaneous rupture of spina bifida.

The Signs and Symptoms of Spina Bifida.—There is a globular or oval swelling which is usually translucent to light. It increases in size on coughing or crying and may or may not pulsate. Occasionally we are able to partially reduce the swelling. A gradual increase in the size of the swelling occurs and in the course of a number of weeks or months ulceration of the skin takes place, rupture of the sac occurs and meningitis usually arises.

The external appearance of the sac gives us definite diagnostic and prognostic information. When the outer covering of the sac is made up of sound skin the diagnosis lies between simple meningocele and syringomyelocele. In these two types of cases there is complete development of the cord and the cutaneous covering, which make them the most favorable types of cases for surgical intervention.

In contrast to the sound skin of the meningocele and syringomyelocele, the outer covering of the meningomyelocele presents an entirely different aspect. It is made up of three zones, the dermatic, the serous and the medullary zones. The medullary zone forms the outer covering of the sac and is made up of pia mater and the unclosed portion of the neural tube. It is this zone which must be preserved in its entirety at the operation and replaced in the spinal canal; otherwise irreparable damage is inflicted on the nerve structures attached to this zone.

In our series of cases operated upon and the studies made on the cases that were not operated upon, we have found meningomyelocele the most common form. In this type there is a partial arrest of development of nerve

structure as well as the mesenchymal defect. The nerve structures and pia mater are adherent to the summit of the sac. As the sac increases in size, traction is exerted on the nerve structures lessening their nutrition and impairing their function. Aside from the question of elongation of the nerve elements, the destructive influence of the fluid pressure on the nerves at the neck of the sac as they emerge from the spinal canal must be considered.*

It is because of these factors, the elongation of the nerves with their subsequent destruction or partial destruction, the damage done by the fluid

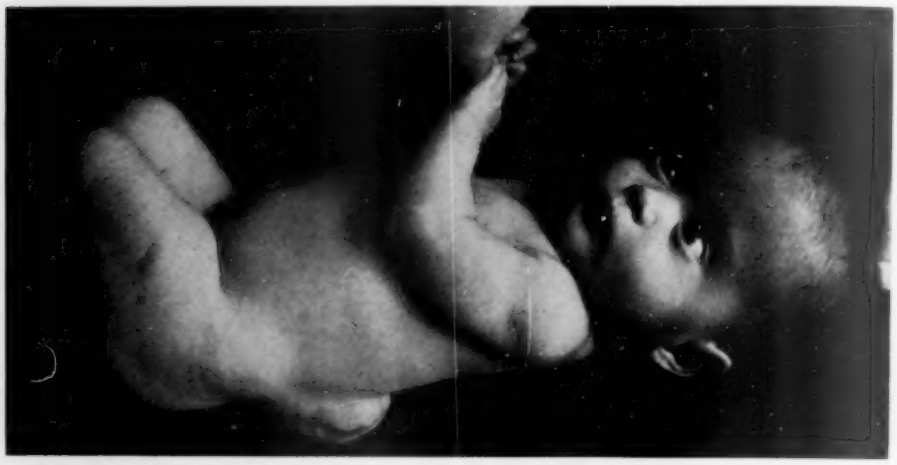


FIG. 4.—Spina bifida associated with hydrocephalus.

pressure on these nerves from within, and the almost certainty of ulceration and secondary meningitis of the vast majority of these cases we are making our plea for early operation.

The Question of Operation.—In recommending operation on these deformities we must take into consideration: 1. The degree of arrest in development of the nerve structures. 2. The damage due to the nerves or the cord after birth. 3. The possibility of hydrocephalus following these operations. 4. The presence of ulceration or infection of the cutaneous wall.

The presence of hydrocephalus associated with spina bifida forbids operation. Incontinence of urine and feces or the presence of gross deformities, such as, flaccidity of the lower extremities (Paraplegia), also forbids operation. It is of interest that in Case II, a child showing flaccidity of the lower extremities, with reactions of degeneration in all the muscles of those extremities, to find that after a spontaneous rupture of the sac, tone appeared in these muscles and there were slight movements on irritation of the plantar surfaces; a fact which indicates that pressure upon and the elongation of the nerves by the increase in the size of the sac and by increase in the amount of fluid explain some of these phenomena.

* These developmental defects occur at a time in fetal life when the spinal cord is the same length as the spinal column. With the growth of the vertebræ the cord apparently ascends consequently in syringomyelocele and meningomyelocele the nerve structures which are fixed outside of the spinal canal are stretched and distorted and their function impaired.

In Case V of this series, an infant referred by Dr. P. B. Bland, showed a spina bifida, also a defect in the muscles of the posterior abdominal wall, an opening into the interior of the bowel, and an intussusception of the entire large intestine and the appendix protruding out of the cleft adjacent to a spina bifida.

The mere presence of club feet, paralysis or atrophy of a group of muscles, should not contraindicate operation. Case XI, age eight months, was observed in our Clinic for one year. In this case the skin over the sac was sound and when the child was first seen, at eight months of age, the swelling was about the size of a tangerine orange. Examination of the extremities showed loss of muscle tone but no paralysis in either leg. The child reported at three month intervals for examination. At the end of the sixth month, when the child was fourteen months of age, the swelling had increased in size. The muscles of the legs were atrophied and power was lessened. At that time we tried to persuade the mother to have the child operated upon, but without avail. At the end of another six months, the sac was greatly increased in size, there was almost complete loss of power in the left leg and decidedly more weakness in the right leg. The mother now gave her consent to the operation. He crawled about the floor six months after the operation. One year after the operation he was able to stand and is now able to walk a few steps. His mother states he has control of the bowels except when he has diarrhœa. He does not have control of the bladder at night but does during the day.

FIG. 5.—Spontaneous rupture of meningomyelocele—note the thickened skin.

This case is of interest because it shows the damage which occurs to the nerve structures because of elongation due to enlargement of the sac and the fluid pressure within the sac; a conclusion verified by the history, by our observation and by the improvement following the operation. The delay in this case was due to the false feeling of security afforded by the sound skin over the sac.

This leads us to the question of the time for operation. In uncomplicated cases it can be expressed in a few words. Before the sac is obviously enlarging and before ulceration has occurred. Enlargement causes traction on the nerves; ulceration prohibits operation because of the practical certainty of meningitis resulting. Unless hydrocephalus is present the fear of it following operation should not be considered. It is a possibility. Meningitis or

nerve destruction is almost a certainty without operation, except in rare cases of spontaneous closures.

The treatment of spina bifida may be said to be the conversion of a congenital decompression of the spinal cord to a surgical decompression, or, when the dura is sutured, a closure of the gap.

Operation.—Position of the patient. The child was placed on the abdomen and a sand bag was laid beneath the hips in order to elevate the hips and to prevent an undue loss of cerebrospinal fluid during the operation. As a rule, with the patient in this position, the only fluid lost will be that from within the sac.

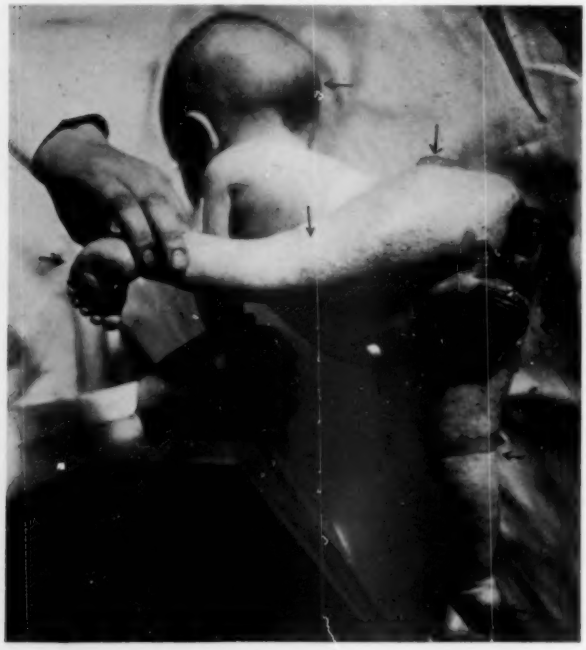


FIG. 6.—Spina bifida associated with bilateral club feet, fibrous ankylosis of both knees, and prolapsus of rectum.

An incision was made through the sound skin encircling the neck of the sac; bleeding was carefully controlled because young children stand loss of blood very badly. The neck of the sac was dissected out carefully, free of the muscles and fascia, down to the opening out from which neck of the sac protruded. The outer covering of sound skin, or in meningomyelocele, the dermic, serous and medullary zones, remained as a cap over the distended sac which was now free at its neck. The cutaneous cap was then reflected backward by careful dissection of the skin from the arachnoid layer, extreme care being taken not to rupture the sac. Little difficulty was encountered during the separation until the medullary zone was reached. At this point it was impossible to further separate the cap. An incision was made separating the medullary zone from the serous and dermatic zones. The medullary zone was left on the distended sac which was now composed of the medullary zone, the arachnoid and the nerve structures. The sac was then opened and collapsed, and care was taken not to permit any bleeding to take place into the collapsed sac. The collapsed sac was placed in the trough of the spinal canal. The question now arose whether the dura should be closed or whether it should be left open and a spinal decompression operation performed. It will be recalled that in the early description of these cases the dura was observed to have been in one of three places; first, there was a cleft in which all the dura remained in the spinal canal and the sac protruded through this opening. Second, the dura was extended through the bony structure upward along the neck of the sac as a cuff reaching about one-third the distance to the fundus. Third, the dura was cleft and blended with the erector spinæ mass group of muscles.

If there is a simple cleft of the dura the sac which consists of the collapsed membranes and nerve structures should be replaced in the spinal canal and the dura closed, or, if the dura extends upward as a cuff the collapsed sac and nerve structures should be replaced in the spinal canal and the dura

closed from within the sac, using enough of the dura in suturing to comfortably accommodate the collapsed sac and its nerve structures. If the dura is blended with the erector spinæ mass it will be impossible to obtain a sufficient amount of dura to make a closure over the collapsed sac. In this type of case a spinal decompression should be done, leaving the dura adherent to the muscle, and using the dura and muscle flap as a protection over the collapsed sac. As a matter of fact spinal decompression should be done on any of the cases if the redundant sac is so large that it precludes closure of the dura without serious encroachment on the nerve structures. The remainder of the operation should be performed either by the method of Babcock or Frazier.

FIG. 7.—Spina bifida associated with intussuscepted bowel through a defect in the posterior abdominal wall.

Post-operative.—The child should be placed on the abdomen, the foot of the bed being elevated in order to prevent any leakage of cerebrospinal fluid. In spite of this precaution we had in two of our cases a small temporary leak lasting for several days.

Conclusions.—Cases of spinal and cranial defect do not require extensive repair to prevent recurrence. Operation should be performed early in life before ulceration of the sac has occurred and before there has been any decided increase in the size of the sac. We have satisfied ourselves that increase in the size of the sac, especially in meningomyelocele, causes degenerative changes in the nerve elements because of traction and malnutrition. Cranial defects are due to unequal development of the cranial bones.

Hydrocephalus associated with spina bifida precludes operation, but the possibility that hydrocephalus might develop after operation should not prove prohibitory.

Paraplegia of the lower extremities, paralytic club feet, incontinence of urine and feces, and gross visceral defects also contraindicate operation.

CASE REPORTS.—CASE II.—D. C., female, age one month, referred to the DaCosta Clinic by Doctor Bauer. On admission a lumbar meningomyelocele was observed. X-ray examination: A spinal defect involving lower lumbar region was reported. On the date of admission there was some ulceration of the sac. The legs were flaccid—reflexes were absent. Neurological examination showed reaction of degeneration in both extremities. Hydrocephalus was not present.

Two days after admission to the ward the sac collapsed. (Fig. 1.) It will be seen from this picture, taken one week after the collapse that there has been some restoration of function to the muscles of the right leg. This fact was borne out by examination of

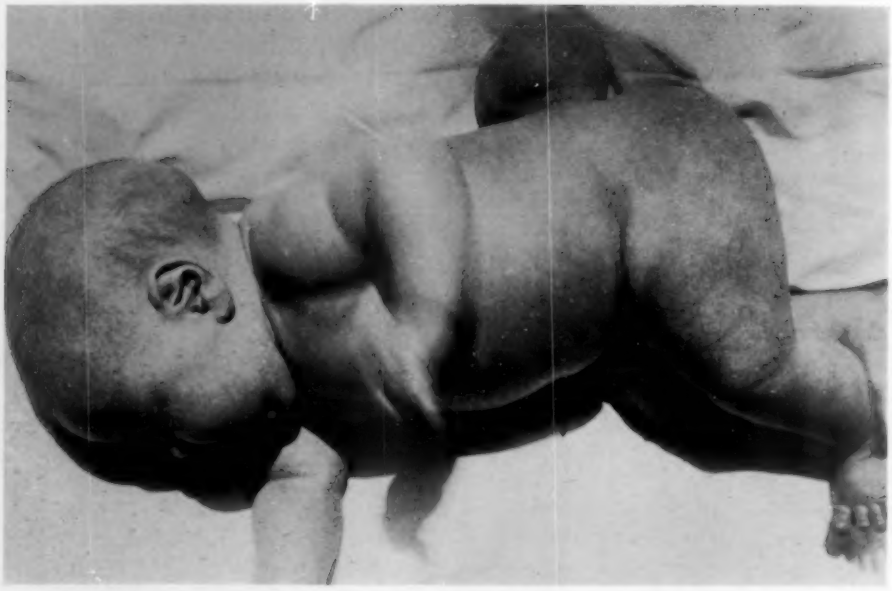


FIG. 8.—Spina bifida—meningomyelocele.

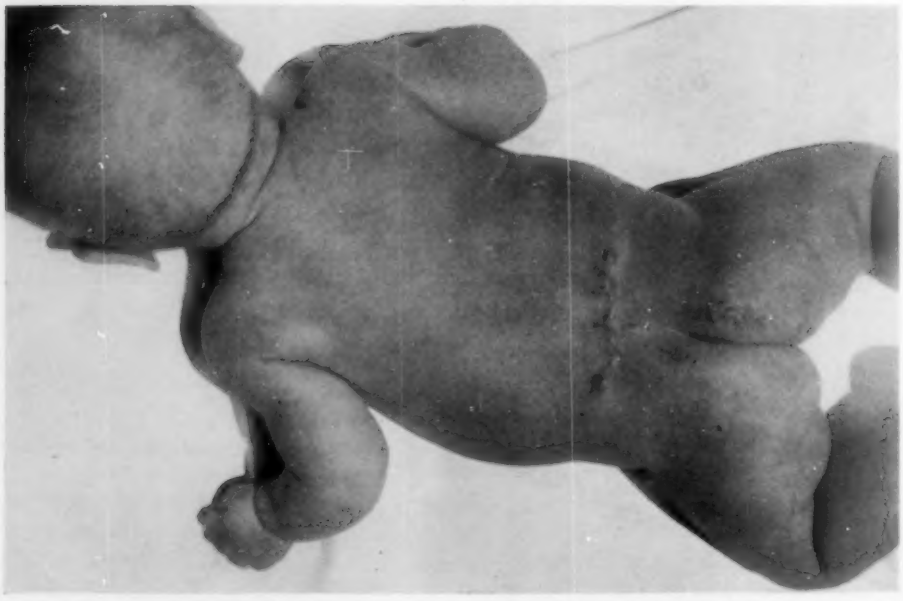


FIG. 9.—Same patient one month after operation.

the extremities. There was a return of muscle tone, and on irritation of the plantar fascia a slight movement of the right foot. Further examination disclosed a defect in arches 6–7–8–9–10 thoracic vertebræ. (Fig. 2.)

Two weeks after the spontaneous rupture of the sac it was noticed that the head was beginning to enlarge. This increase in size was not very noticeable during the first week. The second and third weeks the head was decidedly enlarged. There was wide separation of all the bones of the vault of the skull. (Fig. 3.) There was no return of the sac at this time or at any time up to the death of the child, six weeks after admission.

This case teaches us because of the spontaneous rupture of the sac there was some restoration of function in the legs, therefore, the presence of the sac, either by reason of traction on the nerve elements or fluid pressure on the nerve structures, causes a destructive change to take place in the spinal cord, cauda equina or spinal nerve. Is it not more reasonable to arrest this process of nerve destruction by early operation than to permit it to persist and cause irreparable damage?

This case also demonstrates the association of spina bifida occulta with true spina bifida; and lastly, the enormous increase in pressure manifested by the hydrocephalus (Fig. 3) did not cause a return of the sac which had not been surgically repaired.

Case III.—H. C., female, eight weeks old. (Fig. 4.) Showing the presence of hydrocephalus associated with meningomyelocele. This case was observed in the Clinic for three weeks, the lumbar sac did not increase in proportion to the size of the head. The presence of hydrocephalus contraindicates operation on the spina bifida.

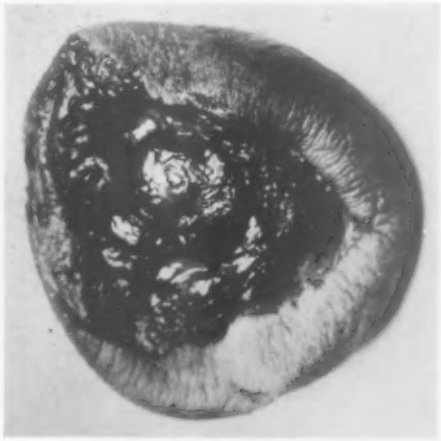


Fig. 10.—Skin and part of the cerebellum from cranial encephalocele.

Case IV.—M. B., male, age two months. At birth a sac was present about the size of an orange. One week after birth the sac collapsed.

Figure 5, taken seven weeks after the spontaneous rupture shows no evidence of recurrence. The protrusion does not contain fluid, it is made up of thickened indurated skin.

In this case the associated deformities of club feet, fibrous ankylosis of knees and prolapse of anal mucous membrane are present. (Fig. 6.) Three months after the discharge of the patient from the hospital, there was no evidence of recurrence of the sac. This child was not operated upon.

Case V.—B. C., female, one week old, was seen by us on the service of Dr. P. B. Bland. (Fig. 7.)

There was lumbo-sacral meningomyelocele present. In addition a defect in the lumbar muscles and the posterior part of the sigmoid. The mass protruding in the large bowel intussuscepted through the bowel and muscle defect. This mass was reducible. The child died one week after birth. No operation was done.

Case VI.—M. Q., female, age three months. History No. P–11169. Transferred from the service of Doctor Bauer.

X-ray examination disclosed a defective first lumbar vertebra. (Fig. 8.) On inspection of the sac the zona medulovasculosa was present (Meningomyelocele). No deformities of the extremities were present, no incontinence of urine or feces, no evidence of hydrocephalus. Operation.—Performed April 30, 1927, as described. Healing was by first intention. The child was discharged on May 30, 1927, in excellent condition. Progress.—Figure 9 shows appearance of back one month after operation. Report of January 24, 1928 (nine months after operation). There has been no evidence of recur-

rence of deformity. She has not been sick a day. She is able to stand and walk a few steps without assistance.

Case VII.—F. G., female, age two months. History No. O–4445. Referred from the service of Dr. P. B. Bland on November 10, 1925. At birth there was noticed a swelling in the occipital region to the right of the midline. The sac increased on crying, was translucent to light. It was partially reducible and was about the size of a duck egg. Operation was performed November 12, 1925, by excision of the sac. The dura did not form any part of the sac but was adherent to the margin of the bone. The

Fig. 11.—Occipital encephalocele.

arachnoid was closed by a continuous catgut stitch. The defect in the bone would admit the tip of the index finger. It was to the right of the midline, between the foramen magnum and occipital protuberance. I believe the opening was the result of delayed development of the occipital bone on the right side. The opening was closed by fascia in which the periosteum adjacent to the opening was included in the bite of the needle. Four sutures were required to close the opening. The scalp was closed without drainage.

Progress.—Patient made an uninterrupted recovery. Sutures were removed from the scalp on the eighth day. Patient was discharged from the hospital, December 31, 1925. Report from the child's mother January 16, 1928 (child two years and four months of age). There has been no sign of return of the lump. She is bright, happy and normal in every way.

Case VIII.—R. M., female, age two months. History No. P–4162. Referred to the Clinic by Doctor Bauer, November 23, 1926. There was a large sac to the left of the midline in the occipital region. The sac was not reducible, it increased in size when the child cried and it was not translucent to light. A distinct pulsation was present. A diagnosis of encephalocele was made. Operation December 4, 1926.—An incision was made encircling the base of the sac through the sound skin. The neck of the sac was exposed by careful dissection. The left lobe of the cerebellum was partially prolapsed through a defect in the occipital bone. The skin flap was reflected backward and separated from part of the cerebellum. At the fundus of the sac, the brain was so adherent, I resected part of the cerebellum with the skin cap. (Fig. 10.) Bleeding was controlled without difficulty. The bone defect was covered with a fascial flap, the sutures passing through the periosteum at the margin of the bony defect. The scalp was closed without drainage.

Progress.—That evening the child's temperature rose to 105, pulse 150. She died the following morning. December 5, 1926. Post-mortem examination showed that there was a twist to the left of the cerebral peduncle. Inspection of the bones of the skull: a foramen is noticed in the occipital bone to the left of the midline; the left temporal and the left part of the occipital bones are smaller than the right temporal and occipital bones. This finding would confirm our opinion that the cause of cranial defect is an unequal development of the cranial bones. The question of increased pressure of the cerebrospinal fluid does not seem to be as potent a factor in the production of cranial defects as some think it to be in the development of spinal anomalies.

Case IX.—B. P., female, age one month. Referred from the service of Dr. P. B.

Fig. 12.—Case of syringomyelocele.

Bland, November 22, 1926. There was a sac protruding through the skull to the left of the median line which was present at birth. (Fig. 11.) The sac increased in size when the child cried. It was opaque to light and there was a distinct pulsation present. The diagnosis of encephalocele was made. Operation December 18, 1926.—It was found that the cerebellum was definitely adherent to the fundus of the sac, and was separated with some difficulty. Again the question of excision of part of the cerebellum, which had been done in previous cases, arose. We decided to replace the cerebellum in the cranial box. The arachnoid was sutured. The bony gap which was about one-half inch in diameter was covered with fascial flap. The scalp was closed without drainage.

Progress.—The child made an uneventful recovery. Discharged from the hospital, December 31, 1926. We have not had a report on this child since April, 1927. The family changed their residence to another city. The last report of April was there was no evidence of a recurrence of the sac. The child appeared normal in every way.

Case X.—M. P., female, age two months. History No. N–488. Admission through the dispensary on June 30, 1924. Examination disclosed a sac in the midline posteriorly in the occipital region which increased when the child cried—was partially reducible and did not pulsate. The skin over the sac was sound. Operation on July 2, 1924.—Excision of sac, closure of opening as described in the preceding case. Closure of the scalp without drainage. Report of January 13, 1928.—Three years after operation, there was no return of the sac. The child was extremely nervous when interviewed, appeared apprehensive and fearful. She had difficulty with her speech. Her general health was excellent.

Case XI.—W. N., male, age two years. History No. N–3175. (Fig. 12.) At birth a small swelling was noticed in the midline posteriorly. It was observed by the mother that the baby seldom moved his feet, as time passed he did not attempt to walk. The left leg was thin and flaccid, the right leg had some muscle tone. When he was about a year old the tumor in the back began to enlarge. His mother noticed that he had less power in his legs. During the second year the sac greatly increased in size. Operation

April 17, 1926.—A syringomyelocele was found. The nerve structures which included the lower portion of the spinal cord, in the centre of which there was a distinct sac, the remainder of the cord was stretched over the sac. The cord with the pia arachnoid were dissected from the skin. The dura surrounded the neck of the sac. The sac collapsed. Because of the redundant membranes and elongated nerve structures, it was impossible to close the dura. A spinal decompression was performed.

Progress.—There was some leakage of cerebrospinal fluid for several days after the operation. One month after the operation the child stood up in his crib. Report of January, 1928, there is no recurrence of the sac. A definite pulsation can be felt at the site of the old defect (spinal decompression). He is able to stand and walks with

FIG. 13.—One month after closure of syringomyelocele.

assistance; he had control of his bowels, except when he has diarrhœa, but not of his bladder at night. He is bright and intelligent. There is no evidence of hydrocephalus.

CASE XII.—Helen S., female, age five months. History No. O-1246. At birth a sac was present at the third lumbar vertebra. The outer surface showed the zona medulovasculosa; it was not ulcerated nor was there any evidence of infection. Operation—September 9, 1925, by excision of the cutaneous cap, the replacement of cord and zona medulovasculosa in the spinal canal, closure of the dura, which encircle the neck of the sac, utilizing all of the dura present to form a wide covering for the cord and membranes, at the site of the defect. The muscles were closed by the Frazier method.

Progress.—Child made an uneventful recovery. Discharged from the hospital, October 1, 1925. We have not been able to trace this case since her discharge. Figure 13 shows closure after operation.

CASE XIII.—E. J., male, two months of age. History No. M-3253, admitted October 25, 1923. X-ray examination showed a defect of 2-3-4-5 lumbar vertebræ, a large meningomyelocele was present. Operation October 28, 1923.—Patient died eight hours after operation; cause of death, operative shock.

CASE XIV.—M. N., female, age two months. History No. M-3531, admitted October 17, 1923. Diagnosis, meningomyelocele, lumbar region. Operation October 20, 1923. Under ether anæsthesia, excision of cutaneous cap, replacement of zona medulovasculosa, arachnoid and nerve structures in spinal canal. Closure of the dura.

Progress.—Patient reacted from the anæsthetic. No evidence of operative shock. Third day developed temperature 100, pulse 120. From third to eighteenth day the child showed progressive signs of meningitis. She died on the eighteenth day from septic meningitis.

CASE XV.—L. S., male, age six months. History No. P-607. Admitted to the hospital, June 15, 1926. Diagnosis, meningomyolocele of the lumbo-sacral region. Operated on June 29, 1926, and died the same day; cause of death, operative shock.

HEART INJURIES—WITH SUTURE*

By Herbert H. Schoenfeld, M.D.

of Washington, D.C.

George Fischer, in 1868, compiled a series of 452 cases of heart injuries, with a 10 per cent. recovery without surgical intervention. Sometime later Block, in 1882, recorded his observations on experimental wounds of the heart in rabbits, which were treated surgically. Billroth, in spite of this, stated in unequivocal terms that "the surgeon who should attempt to suture a wound of the heart would lose the respect of his colleagues." At the eleventh International Medical Conference in Rome, Del Vecchio demonstrated healed wounds of the heart in dogs following suture; and about one year later, in September, 1895, Cappelan of Norway operated the first described case in the human subject. The second was done by Farina of Rome in March, 1896. The third was done by Rehn of Frankfurt in September, 1896. That of Rehn was the only one of the three to recover. In 1908, Vaughn reported a series of 150 cases compiled from the literature, including a second case of his own, which resulted in a successful termination.

I have taken, for the purpose of presentation, the compilation of Peck, in the Annals of Surgery, 1909. Of the cases operated from 1896 until that date, there are 160 cases listed.

Total recoveries	59	36.87 per cent.
Total deaths	101	63.13 per cent.
In O.R.	16	10.00 per cent.
In 24 hours	28	17.50 per cent.
Within 7 days	38	23.75 per cent.
After 7 days	19	11.87 per cent.

The next compilation I have taken is that of Pool, listing 77 cases between 1909 and 1912, published in the Annals of Surgery in 1912.

Total recoveries	42	54.5 per cent.
Total deaths	35	45.5 per cent.
In O.R.	8	10.4 per cent.
In 24 hours	11	14.3 per cent.
Within 7 days	11	14.3 per cent.
After 7 days	5	6.5 per cent.

The third compilation, listing 49 cases from 1912 to 1923, is by Smith, published in the Annals of Surgery in 1923.

Total recoveries	35	71.44 per cent.
Total deaths	14	28.56 per cent.
In O.R.	3	6.12 per cent.
In 24 hours	6	12.24 per cent.
Within 7 days	4	8.16 per cent.
After 7 days	1	2.04 per cent.

* Read before the George Washington University Medical Society, Dec. 17, 1927.

Age of patient Name of surgeon Date	Etiology Location	Time between injury and operation	Drainage	Complications	Sequelæ	Result
Rhodes: 1922: 16 years	Stab wound r. ventricle	About 2 hrs.	No	None	None	Died on table.
Vautrin & Guillemin: 1922	Stab wound l. auricle	1½ hrs.	No	None	None	Recovery.
Klose: 1922: 40 years	Stab wound r. auricle	3 hrs.	Yes	D.T.: Pericarditis: Broncho-pneumonia	None	Died, 18 days.
Rhodes: 1923: 23 years	Stab wound r. auricle	About 1 hr.	No	Pericarditis: Pleurisy: Ac. Tonsillitis. Etc.	Few pleural adhesions	Recovery.
Drummond: 1924: 25 years	Stab wound l. auricle	2 hrs.	No	Pneumothorax: pleural effusion	None	Recovery.
Maguire: 1924: 18 years	Stab wound r. auricle	About 2 hrs.	Yes	Pleural effusion	None	Recovery.
Meyer & Brams: 1924: 37 years	Gunshot wound l. ventricle	About 6 hrs.	No	None	Slight sinus arrhythmia	Recovery.
Brocq: 1924: 35 years	Stab wound l. ventricle	About 1 hr.	No	Heart stopped on table; started with massage	None	Recovery.
Comolli: 1924: 38 years	Stab wound l. ventricle	4 hrs.	Yes	None	None	Died, 5 hours.
Grove, Fernandez & Grove: (6 cases): 1924 #1. 23 years	l. ventricle	Immediate operation	No	None	None	Died, 4 hours.
#2. 30 years	Stab wound l. ventricle	Not stated	No	None	None	Died, 15 days.

#3. 20 years	Stab wound l. auricle	?	Yes	None	None	Recovery.
#4. 18 years	Stab wound l. ventricle	?	No	None	None	Recovery.
#5. 20 years	Stab wound l. ventricle	?	No	Purulent pericarditis	None	Died, 25 days.
#6. 27 years	Stab wound l. ventricle	?	No	None	None	Recovery.
Long (2 cases): 1925: #1. 32 years	Knife: left auricle	?	Yes	Serous effusion	None	Recovery.
#2. 45 years	Stab wound l. ventricle	?	No	None	None	Recovery.
Schoenfeld: 1925: 5 years	Stab wound l. ventricle	About 3 hrs.	No	None	None	Recovery.
Brocq: 1925: 24 years	Gunshot wound l. ventricle	About 3 hrs.	No	None	None	Died, 48 hours.
Flach: 1925:	Knife: l. ventricle	1 hr. or less	Yes	Pneumonia	None	Died, 5 days.
Otto: 1925: 24 years	Stab wound r. ventricle	?	No	Osteomyelitis	None	Recovery.
Lenormant: 1926	Stab: r. ventricle	About 2 hrs.	No	Pericarditis	None	Recovery.
Duschl: 1926	Stab: r. ventricle	?	No	None	Incapacitated	Recovery.
Davenport: 1920: 44 years	Stab wound r. ventricle	About 1½ hrs.	Yes	None	None	Recovery.
Hesse: 1921: 40 years	Gunshot wound l. ventricle	2 hrs.	No	None	None	Died, 2 days.

We have examined the literature, and the individual cases reported in the literature from 1923 to 1926, finding 25 cases, including our own. (See Table.)

Total recoveries	16	64.0 per cent.
Total deaths	9	36.0 per cent.
In O.R.	1	4.0 per cent.
In 24 hours	2	8.0 per cent.
Within 7 days	3	12.0 per cent.
After 7 days	3	12.0 per cent.

The majority of injuries of the heart reported in the literature are stab wounds, usually with sharp instruments, and, less frequently, are due to gunshot wounds.

Death most frequently occurs very promptly, due to shock and hemorrhage, before any treatment can be instituted. The classical symptoms described many years ago include an anxious expression with restlessness; the patient may be unconscious; there is marked shock with pallor and cyanosis; the skin is covered with cold sweat; there is usually dyspnœa, the pulse being feeble and fast.

The physical examination shows a wound over the precordium which frequently does not bleed extensively. There may be evidence of pneumothorax or hæmo-pneumothorax. The precordial area of dulness may or may not be increased in size. The apex impulse is altered. The heart sounds are usually distant.

The diagnosis of cardiac injuries is not always easy, the diagnosis being made upon symptoms which may sometimes be very readily confused with, and present an almost identical picture to that of pulmonary, pleural, and pericardial injuries.

The surgical treatment should be prompt, even in the face of many usually definite contra-indications to surgical intervention. It is felt that the gravity of heart injuries demands exploration, because, as may be seen by the figures presented above, prompt intervention has reduced the mortality of heart injuries from about 63 per cent. reported in the first series in 1909, to approximately 30 per cent. at this time.

Time does not offer for the presentation of the types of operation which may be performed, but the statement should be made that surgery should be carried out early, and that generally speaking, better results have been obtained by complete closure without drainage. Drainage lends more possibility of infection, and infection is the principal factor, along with bronchopneumonia, in the late mortality of these patients.

The repair of heart injuries is generally conceded to be carried on in the same fashion as the healing of other tissues; namely, by the laying down of fibrous tissue. Heart muscle does not regenerate in these cases. Around the area of scar there is, however, described an area of hypertrophy of the muscle cells.

CASE REPORT.—Melvin Jones, aged five years, was admitted to the Children's Hospital about 2 P.M. on September 20, 1925, with a history of having run into or fallen upon a pair of scissors at about 12.30 P.M.

At the time of admission, he was in profound shock, had a marked pallor, some cyanosis, the extremities being cold and clammy, and the body bathed in cold sweat. The pulse was hardly perceptible, and there was a puncture wound in the fourth left costal interspace, about one inch from the edge of the sternum. The left chest was hyper-resonant, particularly over the cardiac area, to beyond the left nipple line. The heart sounds could not be heard, and there was bronchial breathing over the cardiac area.

The child was given normal salt solution by hypodermoclysis, immediately, and about sixteen minims of fibrogen, following which the pulse picked up slightly.

An X-ray made at this time showed the heart normal in size and position; the lung field entirely clear. There was no evidence of pneumothorax. The lung was in no way retracted, and there was no evidence of fluid.

At about 3.40 P.M., under ethylene-oxygen anæsthesia, an incision was made through the superficial tissues over the third rib, from the costo-chondral junction to the chondro-sternal junction. A similar incision was made above the fifth rib, and the flap of skin and muscle was turned laterally. The third and fourth ribs were cut through at the costo-chondral and chondro-sternal junctions, the third and fifth intercostal muscles being sectioned transversely and at the sternum, and the entire flap was turned laterally. Following the control of hemorrhage from the internal mammary artery, the pericardium was opened transversely, releasing a large blood clot, and some free blood, from the pericardium. (It is interesting to note that on opening the chest the pericardium had dropped away from the thoracic wall.) The heart, as well as the pericardium and superficial tissues, showed a small puncture, one-quarter inch in length, extending longitudinally on the anterior surface of the left ventricle about one and one-half inches above the apex, from which, on contraction, a stream of blood was ejected about sixteen to eighteen inches. This wound was closed with two interrupted silk sutures, following which the pericardium was closed, and the superficial tissues were sutured without drainage. During the closure, high pressure was exerted by the anæsthetist with his closed anæsthesia for the purpose of dilating the collapsed lung caused by pleural injury. A large flat dressing was applied over the wound, and an adhesive dressing applied to the left chest. At the conclusion of the operation, the pulse was quite perceptible and of good force. A transfusion of whole blood was not easily done, but about a hundred c.c. of blood, which could not be infused, was given per rectum.

The day following the operation, the patient was greatly improved in every way. He was seen in consultation with Doctor Wall before leaving the hospital, who reported the chest examination normal except for slight bulging in the lower left chest anteriorly; a diffuse, poorly defined apex beat, one and one-quarter inches below the nipple; and a slight decrease in the intensity of the respiratory sounds in the left chest. The convalescence was steadily progressive up to the time of his discharge from the hospital, October 29, 1925.

On December 7, 1927, Dr. Thomas S. Lee, of Washington, very kindly consented to give the child a thorough cardio-vascular examination, which showed the chest symmetrical, except for the operative scar. Expansion was good and even on both sides; no visible pulsation; moderate epigastric pulsation when recumbent. The apex beat was barely palpable in the fifth intercostal space, within the midclavicular line. The size of the heart was normal, the teleröntgenogram showing the transverse diameter of the heart to be 8 cm., and that of the chest 19½ cm. The aortic arch was not enlarged. There were no murmurs or other signs of pericarditis or endocarditis. The myocardium was in satisfactory condition, as was shown by the quality of the cardiac tones, the normal response to moderate exercise tests, and the normal electro-cardiogram. The heart rate, sitting, was 100, the patient being somewhat nervous under examination. There was a moderate degree of sinus arrhythmia, which is of no clinical significance

in a child. The blood pressure was systolic 100 mm., diastolic 60 mm., in both arms. There was no sign of circulatory inefficiency in the lungs, the liver, or elsewhere.

BIBLIOGRAPHY

Vaughn: Annals of Surgery, 1909.
Peck: Annals of Surgery, 1909.
Pool: Annals of Surgery, 1912.
Smith: Annals of Surgery, 1923.
Vautrin et Guillemin: Rev. Med. de l'est, Nancy, 1923.
Vidal: Bull. et Mem., Soc. de Chir. de Par., 1923.
Hesse: Deutsche Ztschr. f. Chir., Leipz., 1923.
Klose: Arch. f. Klin. Chir., Berl., 1923.
Rhodes: Tr. South. Surg. Assn., Phila., 1924.
Comolli: Sperimentale Firenze, 1924.
Davenport: J. Am. M. Assn., 1924.
Grove, Fernandez, and Grove: Cong. Nac. de Cirug., Valparaiso, 1924.
Drummond: Brit. J. Surg., Bristol, 1925.
Flach: Soc. de Med. Mil. Franc., Bull. Mens., Par., 1925.
Long: Bost. Med. and Surg. J., 1925.
Maguire: Surg., Gyn. and Obst., 1925.
Meyer and Brams: Ill. Med. J., Oak Park, 1925.
Otto: Munchen Med. Wchnschr., 1925.
Rhodes: Annals of Surgery, 1925.
Beck: Ann. Med. Hist., N. Y., 1926.
Lenormant: Bull. et Mem., Soc. Nat. de Chir., Par., 1926.
Duschl: Munchen Med. Wchnschr., 1926.
Brocq: Bull. et Mem., Soc. Nat. de Chir., Par., 1926.

PRIMARY HYPERNEPHROMA OF THE LIVER*

By Irvin Abell, M.D.
of Louisville, Ky.

The observation of hypernephroma as a secondary or metastatic deposit in the liver is a common one, its definite identification as a primary growth therein is a rare one.

The existence of accessory suprarenal capsules or adrenal cell nests disseminated at more or less distant points from the chief gland was more or less discredited until Morgagni (1740) confirmed it by his personal observations. The first instances of accessory adrenal tissue were reported by Piccolominus (Peperé) in 1856, since which time various authors have reported them in sundry portions of the urogenital tract and in the abdominal viscera. Bothe gives the following organs and tissues tabulated by Broman, in which adrenal cell nests have been found:

1. *In the male:*
   In the rete testis and epididymis.
   In the paradidymis.
   On the spermatic cord, in the inguinal canal, and above and below the same.
2. *In the female:*
   In the ovary, where they may be mistaken for shrunken corpora lutea.
   On the tubes.
3. *In both sexes:*
   In the retroperitoneal tissue below the poles of the kidneys.
   Along the internal spermatic and ovarian veins.
   On the iliopsoas muscle at the brim of the pelvis.
   At the sacro-iliac synchondrosis.
   In the capsule of the kidney and in the kidney substance.
   On the wall of the neighboring vessels.
   In the solar and sympathetic plexuses.
   Between the transverse colon and the spleen.
   In the right lobe of the liver.
   In the pancreas.

Schmorl, in 1891, reported finding adrenal cell nests in the liver four times in 510 autopsies. Beer in 1904, reported the examination of 150 livers for suprarenal inclusions, in six of which adrenal cell nests were found without any evidence of tumor formation. In five of the cases the inclusions lay partly in and partly just beneath the liver capsule. In one case the inclusion lay in the parenchyma and was separated from Glisson's capsule by an acinus. The suprarenal inclusion was not adherent to the liver in any of the cases and there was no inflammation. In all of the cases the inclusions were in the right lobe near the suprarenal impression. All the subjects were adults and in all the microscopic picture was typical of suprarenal tissue. The high proportion

* Read before Southern Surgical Association meeting, December 14, 1927, Augusta, Georgia.

of inclusions found by Beer will not hold good in general, but it shows the importance of making an examination for suprarenal tissue in any primary tumor of the liver, particularly when not originating in the liver cell.

The embryological explanation of such inclusions is made clear by the work of a number of authors, the report of one of which, Bothe, is in part as follows: "The original studies were made with the hope of obtaining cross-sections of embryos at an age when the anlage of the cortical substance of the suprarenal gland and metanephros are to be found at the same level. Of the specimens studied, including pig embryos of 7, 12 and 19 mm., and human embryos of 7, 12 and 16 mm., respectively, the 16 mm. human embryo alone revealed the two anlages in close proximity. In this specimen cross-sections made at the upper pole of the mesonephros disclosed the presence of the component cells of this structure together with the cortical substance of the suprarenal gland in the closest proximity and at a fairly early developmental stage. The illustration (Fig. 1), which is a modification of the one presented by Wilson and Willis, shows the approximate level at which these sections were cut, namely at the level of the upper pole of the mesonephros. The illustration (Fig. 2) shows the relation of the respective anlagen in cross-section at this level. In describing the section and taking the aorta as a fixed point we find ventrolaterally to it a collection of cells in the mesenchyme representing the cortical substance of the suprarenal gland. The identification of the latter structure is dependent upon the characteristic cortical cells, with a somewhat irregular outline and a large nucleus. Scattered between these cells, are imperfectly formed blood sinuses which are identified by the presence of endothelial cells. The arrangement of the above described cells into strands is the typical finding in the adrenal anlagen. Ventrolateral to these suprarenal cells is the mesonephros and genital ridge, with the mesonephros placed in a lateral position. In this section, as is shown by the illustration, it is difficult to determine the limits of the cortical cells: there is no definite limiting tissue. When studied under high magnification a very narrow band made up of young connective-tissue cells is deflected separating these two anlagen, but the separation is in places very poorly defined, being effected by only one or two connective-tissue cells. The anlagal cells of the adrenal are so situated with respect to the liver, kidney, ovary, testicle, epididymis and

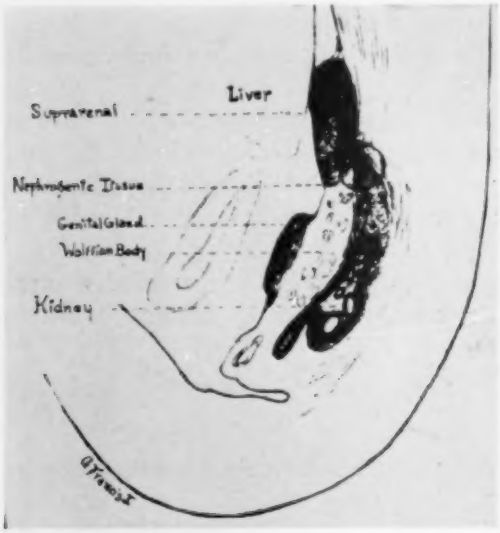


FIG. 1.—See text.

uterus that one can easily see the possibility of adrenal cell inclusions in these organs."

Accepting the theory of Grawitz that hypernephromata are developed from adrenal cell nests the explanation for their occurrence in the liver is at hand. All of the suprarenal inclusions reported by Beer were in the right lobe near the suprarenal impression. In the cases of primary hypernephroma in the liver found in the literature reported in sufficient detail to determine localization seven (de Vecchi, Donati, de Ahna, Hirschler, Cirio, Williams, Swenson) were in the right lobe, one (Peperé) in the left lobe, and two (Starr, Harrigan) in the folds of the falciform ligament, all being in adults. Of the ten cases the tumor was discovered at autopsy in three, two were explored surgically and later recovered at autopsy, while five were surgically removed with recovery of patients. Of the eight occurring in the liver practically all are described as more or less encapsulated tumors situated at the lower border of the liver, some immediately under Glisson's capsule, others completely covered with liver tissue. De Ahna states that fetal inclusions of suprarenal tissue in the liver are generally found on the lower surface of the right lobe: generally they are just beneath Glisson's capsule, but they may be pushed deeper into the parenchyma by the growth of the liver cells and become surrounded on all sides by liver tissue. Here they may remain quiescent, atrophy from pressure or give rise to tumor formation. It is suggested by Hirschler that the reason they are so rarely found in the liver, in comparison with other locations, is that the liver tissue crushes them out entirely as they do not have a very active capacity for growth. The microscopical picture does not differ from hypernephroma originating elsewhere and in the primary deposits there is no involvement of liver cells other than atrophy from compression.

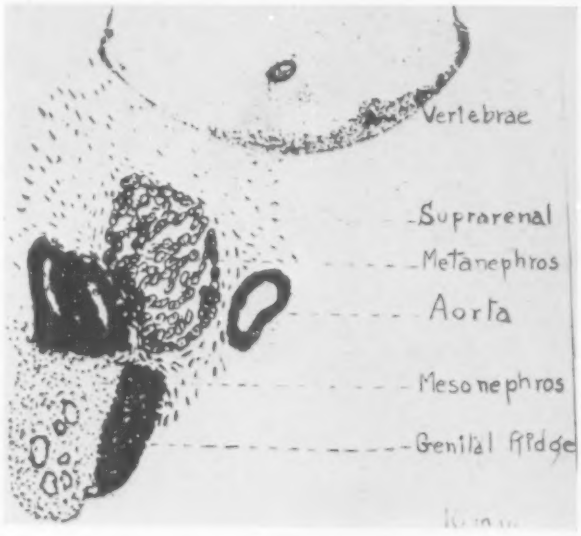


FIG. 2.—See text.

The writer desires to add to the above mentioned cases a report of one of like character coming under his observation.

CASE REPORT.—A. G., female, white, age thirteen months, an only child, came under observation June 16, 1926. Its mother had noted an enlargement in the abdomen one month before and stated that the latter had increased rapidly in size. No change in the child's health had been observed; it was playful, had a good appetite and slept well.

No evidence of discomfort on urination and the character of urine as observed on the napkins had shown nothing unusual. Examination showed a well-developed, well-nourished baby weighing nineteen pounds, with negative findings other than a large rounded mass filling the right half of the abdomen and extending to the left of the midline. While movable, it filled the abdominal cavity to such an extent that no definite conclusion as to its attachment could be reached. X-ray films revealed nothing further than a large rounded tumor. A tentative diagnosis of a right renal embryoma was made and exploration advised. Abdomen opened June 22, 1926, by incision in right linea semilunaris extending from costal margin to a point above Poupart's ligament. Tumor found to originate in the liver being oblong and rounded, approximately five inches in its longest diameter and three inches in its shortest. It was seemingly distinctly encapsulated and connected with the right lobe of the liver by a compressible amount of liver tissue, its attachment corresponding to the entire anterior border of the right lobe extending from the tip of the twelfth rib to a point slightly to left of the umbilicus. The liver tissue was compressed between two rubber covered stomach clamps and the tumor cut away. A hæmostatic running suture of mattress type was then passed through liver tissue distal to clamps. The cut liver edges were brought together by a running suture of catgut. The remaining portion of liver was negative to palpation and inspection: the glands along its lower border showed definite enlargement. Kidneys, adrenals, spleen and remainder of abdominal organs were negative. The patient made an uneventful recovery and at the last examination, November 10, 1927, seventeen months after operation, she presented no evidence of recurrence. She was at that time thirty months of age, weighed thirty pounds, and was well nourished. The abdomen showed the right rectus scar in good condition; no rigidity; no masses.

FIG. 3.—Free surface—130 x 90 x 60 mm. Weight 460 gms.

The tumor was examined by Professor Graves of the Department of Pathology, Medical School of the University of Louisville, whose report is as follows:

PATHOLOGICAL REPORT.—*Gross Description.*—Specimen consists of fairly firm, encapsulated, oval, reddish-gray mass, 130 x 90 x 60 mm., weighing 460 gms. and faintly lobulated. Cut surface is brownish-gray, lobulated and moist. Inner tissue is very friable.

Microscopical Description.—Sections from different portions of tumor show a delicate neoplasm, mostly sharply circumscribed with fibrous tissue, but in places tongues of tumor tissue seem to be infiltrating between lobules of surrounding liver tissue. In the neoplasm itself are fairly numerous large, thin-walled blood-vessels with tumor cells directly adjacent. The tumor cells tend to be arranged in lobules of uneven size and shape, separated by thin, interlacing bundles of fibrous tissue. Otherwise there is very little stroma and individual tumor cells lie in close contact. These cells are rounded or polyhedral with delicate, distinct outlines. The nuclei are small, rounded and tend to lie central. The cytoplasm is scanty, pink and finely granular. The delicate, lace-like appearance

under low power magnification, as well as the detailed appearance of the cells under high power, seems identical with that of hypernephroma. Study of the individual cells reveals no pigment. Mitoses seem absent. There are no structures resembling bile ducts. *Microscopical Diagnosis.*—Hypernephroma. Figures 3, 4, 5, 6, 7.

SYNOPSIS OF HISTORIES OF REPORTED CASES

1. A. Peperé. 1902. Female patient admitted to hospital in grave condition, pleurisy, evening fever, rapidly increasing ascites, which on paracentesis showed a hemorrhagic character. Liver showed enlargement with irregularity of its surface. Clinical diagnosis—malignant neoplasm of liver, probably sarcoma. Autopsy: The surface of the liver was rendered unequal by many yellowish-white nodules of neoplastic appearance, the left lobe being entirely transformed into a neoplastic mass. Spleen, kidneys, adrenals, pancreas, uterus, ovaries, peritoneum, retroperitoneal and mesenteric glands negative for evidence of tumor. The lungs were studded with yellowish-gray nodules the size of a pea. No further metastases found. Histologic examination showed the tumor to be hypernephroma. Peperé calls attention to this case as the first one of primary hepatic tumor arising from aberrant rests of the suprarenal capsule to be described.

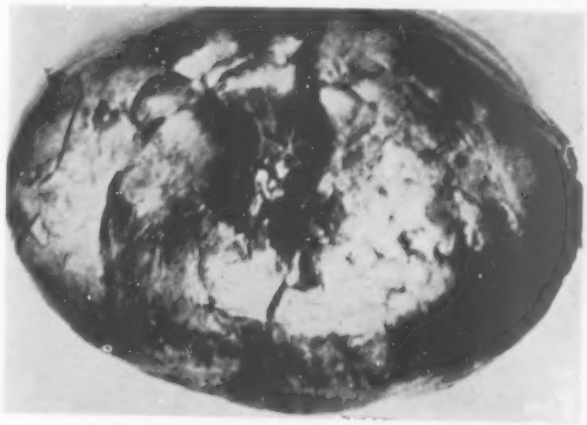


FIG. 4.—Attached surface—130 x 90 x 60 mm. Weight 460 gms.

2. B. de Vecchi. 1904. Female, age twenty-nine, died of puerperal sepsis. A routine autopsy was done, during the course of which a "walnut-sized" tumor was found in the right lobe of the liver above the suprarenal impression. The tumor was entirely surrounded by liver tissue. There was no definite capsule as the boundary was formed by a fusion of the coverings of different lobules of the liver, the adjacent liver cells being atrophied and compressed. There were no further lobules in the liver or elsewhere. Microscopical examination showed hypernephroma. The literature of the subject is reviewed and the view expressed that suprarenal inclusions are probably carried to the liver by the circulation.

3. M. Donati. 1905. Female, age thirty-six, with history of movable tumor in right half of abdomen for six months. The tumor had recently become sensitive and painful, and at time of admission to hospital patient had high fever and presented a sensitive mass movable with liver on respiration. Operation, with tentative diagnosis of suppurative cholecystitis, revealed a tumor of the right lobe of the liver which was removed after placing an elastic ligature around its base. It weighed 370 grams and was covered with a layer of liver tissue 1.2 to 1 cm. thick. The patient recovered. Microscopical examination revealed hypernephroma. The author is inclined to agree with Peperé's theory that the inclusions are carried to the liver by the circulation.

4. M. Hirschler. 1912. The author gives abstracts of cases of hypernephroma of the liver by Schmorl, Kaufman, Peperé and de Vecchi, all autopsy findings, and which he says are the only cases in human beings described in the literature. His own case was in a man of fifty-two dying of diabetes and tuberculosis. The liver tumor had caused no symptoms during life and had not been diagnosed. It occupied almost half of the right lobe of the liver and was almost the size of a "child's head," being 11 cm. in

diameter. The surface of the tumor nearest Glisson's capsule was covered with 1 cm. of liver tissue. The capsule around the tumor was intact except in a few places at which actively proliferating tumor tissue had broken through it. The metastatic nodules in the surrounding liver parenchyma were just like the chief tumor. The liver tissue around the tumor was compressed, the cells small, irregularly shaped, atrophic and many of them filled with yellowish-brown pigment. Microscopical diagnosis: hypernephroma. He cites other cases in which suprarenal inclusions have been found buried in liver parenchyma and is inclined to disagree with Peperé's theory that such inclusions are carried by circulation and to agree with Schmorl's theory that liver tissue gradually grows around deposits of suprarenal tissue on its lower surface and may push them deep into the parenchyma.

FIG. 5.—Cut surface, brownish-gray, lobulated, moist.

5. De Ahna. 1912. Male, age forty-five, with a history of pain in the right side of abdomen for eight weeks. There was a large tumor in the right flank which moved downward with respiration and appeared beneath the costal arch. On palpation it could be pushed upward under the ribs. The left kidney region was free: the right kidney urine showed pus, the left none. Operation by Professor Hildebrand, tentative diagnosis, renal hypernephroma, revealed the tumor to originate in the right lobe of the liver. It was enucleated and its bed closed with suture. Patient recovered. The tumor was the size of a "child's head" and everywhere except where it had been attached to the liver was covered by a thickened Glisson's capsule. Microscopical diagnosis: hypernephroma. The author discusses suprarenal inclusions, refers to Hirschler's report (*vide supra*) published the same year and mentions that he was unable to find any case in literature analogous to this one, since it was the first operation reported for hypernephroma of the liver.

6. F. N. G. Starr. 1917. Female, age thirty-five, presented a mass most prominent in the epigastric region, extending from the costal margin above to about four inches below the level of the umbilicus. Pre-operative diagnosis lay between—(a) an atypical pancreatic cyst, (b) an hydatid of the liver, and (c) a collection of fluid in the lesser cavity. At operation a tumor weighing eight and one-half pounds was found between the layers of the falciform ligament of the liver and removed without rupture. Patient recovered. Microscopical diagnosis: hypernephroma.

7. Carl G. Swenson. 1917. Female, age fifty-eight, ill one year with history of "lump" in epigastrium for seven months. At operation tumor found to arise in the right lobe of the liver, requiring an excision of 2 by 10 cm. of the latter for complete enucleation. Patient recovered from the operation with recurrence of the growth eleven months later. The diameter of the tumor measured 12.5 cm. and it weighed 2 kilograms. Microscopical diagnosis: hypernephroma.

8. A. H. Harrigan. 1917. Female, age thirty-five, with history of severe pain in right upper quadrant of abdomen during two years. Diagnosis: chronic appendicitis and possibly cholecystitis. Operation: appendix removed—gall-bladder and biliary ducts negative for stone. A "walnut-sized" tumor was removed from the falciform ligament

close to the free border of the liver. Closure with suture. Patient recovered. Microscopical diagnosis: hypernephroma. Embryological explanation is discussed as is also the theories of Wilson, Growitz and Stoerch regarding hypernephroma.

9. L. Cirio. 1922. Autopsy report of twenty-eight-year-old man dying after exploratory laparotomy for abdominal tumor. Nodular tumor of the size of the "head of a fetus" attached to the free border of the liver to the left of the gall-bladder which had devoloped outside the liver toward the transverse mesocolon and the hepatoduodenal ligament. Many nodes scattered irregularly in the liver which showed the same appearance as the chief tumor. There were no new growths in any of the other organs; the suprarenal bodies were flattened and intensely pigmented. The nodes were as a rule encapsulated, the liver tissue around the tumor was compressed but with no signs of new growth of the liver tissue itself. Where the capsule was lacking there was no boundary between the liver tissue and tumor tissue, the cells of which showed an active tendency to proliferation. Microscopical diagnosis: primary tumor of the liver derived from inclusions of suprarenal cortex.

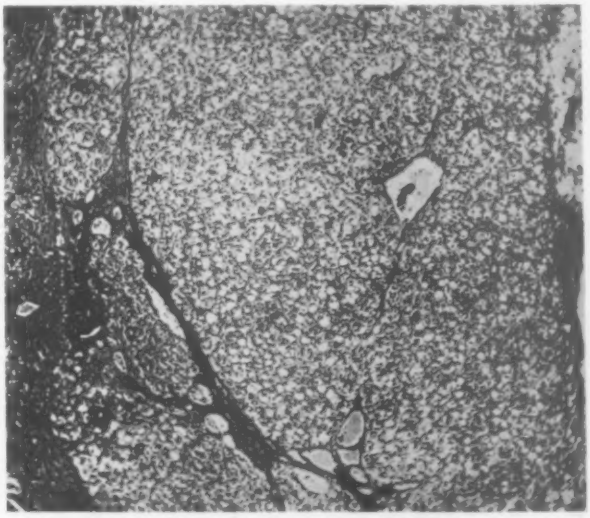


FIG. 6.—Low power microphotograph.

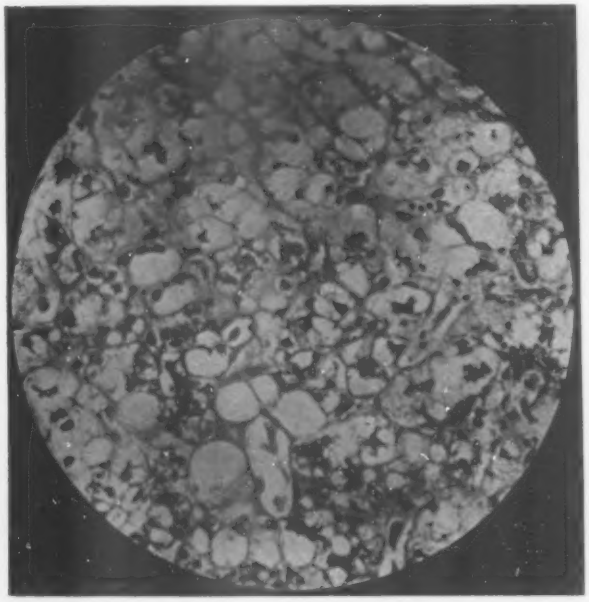


FIG. 7.—High power microphotograph.

10. L. Williams. 1923. Female, age sixty-eight, with a history of acute pain in abdomen two years previous to admission to hospital, at which time a mass was discovered in the right side of the abdomen, apparently in the region of the kidney. At time of admission neuritis of right side of chest and right shoulder, difficulty in breathing and a sense of fulness in epigastrium were noted. The liver was palpably enlarged and the mass below and behind the liver appeared to be about the same size as on previous examination. Exploratory operation by Dr. Carl B. Davis. Liver found to be studded with tumor nodules, one of which was removed. Microscopical examination disclosed a hypernephroma. The patient lived seven months and

autopsy revealed a primary hypernephroma of the liver with extension and metastases in the remainder of the organ. The tumor tissue was found limited entirely to the liver, no metastases being found outside this viscus.

BIBLIOGRAPHY

Peperé, A.: Tumeur primitive du foie originaire des germes aberrants de la capsule serrènale. Arch. de méd. exper. et d'anat. path., Par., 1902, vol. xiv, pp. 765–808.

de Vecchi, B.: Ueber einen Fall von Hypernephrom der Leber. Virchow's Arch. f. path. Anat. (etc.), Berl., 1904, vol. cxxvii, pp. 133–150.

Beer, E.: Ueber Nebennierenkeime in der Leber. Ztschr. f. Heilk., Wien. u. Leipz., 1904, vol. xxv, pp. 381–388.

Donati, M.: Ipernefroma maligno. Arch. per le sc. med., Torino, 1905, vol. xxix, pp. 154–163.

Hirschler, M.: Ein Nebennierentumor in der Leber. Frankfurt Ztschr. f. Path., Wiesb., 1912, vol. ix, pp. 343–361.

de Ahna: Ueber einen Fall von Hypernephrom der Leber, geheilt durch Resektion. Charité-Ann., Berl., 1912, vol. xxxvi, pp. 390–393.

Starr, F. N. G.: Hypernephroma of the Folds of the Falciform Ligament of the Liver. Ann'ls of Surgery, Phila., 1917, vol. lxvi, pp. 318–320.

Swenson, C. G.: Hypernephroma of the Liver. Surg., Gynec. and Obst., Chicago, 1917, vol. xxv, pp. 570–571.

Harrigan, A. H.: Hypernephroma of the Falciform Ligament of the Liver. Annals of Surgery, Phila., 1918, vol. lxviii, p. 395.

Cirio, L.: Sopra un tumore primitivo del fegato di probabile origine cortico-surrenale. Pathologica, Geneva, 1922, vol. xiv, pp. 197–207.

Williams, L.: A Primary Hypernephroma of the Liver. Tr. Chicago Path. Soc., vol. xii, pp. 90–92.

Schmorl: Zeigler Beitr., 1891, vol. ix, p. 524.

Bothe, Albert E.: Hypernephromata. Annals of Surgery, July, 1926, pp. 57–88.

Garceau: Tumors of the Kidney, 1909.

REMOTE RESULTS OF BILIARY SURGERY*

By F. Gregory Connell, M.D.
of Oshkosh, Wis.

Our knowledge of disease processes has been broadened by a study of so-called "Inaugural symptoms" (the monument left to us by the late Sir James Mackenzie), likewise by experiences met with in the recently popularized Periodic Examination of the Apparently Healthy, but definite concrete information relative to the value of certain lines of treatment, or management, call for: 1. Information as to the natural history of untreated disease. 2. Knowledge as to the immediate result of treatment: (a) Relief of symptoms, (b) mortality. 3. Knowledge as to the remote result of treatment. (a) Permanent cure, (b) return of symptoms.

1. The natural history of untreated disease has been given very little serious consideration: but with social services extending from our hospitals to industries, life insurance companies, schools and communities, accurate information will be forthcoming.

2. The immediate result strikes one in the face, is dramatic, has been given much attention; with the mortality rate assuming the most important aspect and relief of symptoms being often mistaken for cure.

3. Remote results, in contrast, are prosaic, many times are not enquired into and frequently too much, usually on the favorable side, is taken for granted; they have been given comparatively little serious study; but, of late, are causing interest which is a natural corollary to the enthusiasm with which the beginnings of disease are being searched for.

In my personal records from 1910 to 1920 I find 336 cases of biliary tract disease of which 150 were not operated upon and 186 were treated surgically. Of those surgically treated, 33 are dead; in 54 the result is unknown, leaving 99 cases in which we are familiar with the post-operative history.

Of the 33 deaths, 4 deaths were remote and unrelated, 2.1 per cent.; 12 deaths were secondary (three months or more after operation), 6.4 per cent.); 17 deaths were primary, 9.1 per cent.; total, 33 deaths, 17.6 per cent.

Of the 17 primary deaths, two followed simple exploratory incisions for malignant disease of the liver; subtracting these two cases leaves 184 cases with 15 primary deaths, that is 8 per cent.

The causes of the 15 primary deaths were recorded as peritonitis, 3; sepsis, 1; sepsis and jaundice, 2; acute hemorrhagic pancreatitis, 1; kidney stone, 1; nephritis, 1; pneumonia, 2; myocarditis, 4; usually not due to method of operating or presence or absence of calculi and will not be considered under this title.

---

* Read before the Western Surgical Association, December 9, 1927.

The 12 secondary deaths were: Cancer of stomach, 3; colon, 2; kidney hypernephroma, 1; myocarditis, 3.

The 4 remote, or unrelated, deaths were: myocarditis, 1; pulmonary tuberculosis, 1; cerebral hemorrhage, 1; railroad accident, 1.

It is of interest to note that of the 33 deaths, carcinoma was a factor in 15 instances (stomach 5, liver 5, colon 2, pancreas 2, kidney 1).

It is to the question of *remote* results, permanent cure or return of symptoms, rather than immediate results, relief of symptoms or death, that I would like to direct attention; no case in this discussion was operated on later than 1920, allowing a fair estimate as to really remote results.

Of these 99 cases with known result, 40 (40.4 per cent.) had a return of symptoms but subsequent developments showed in 15 cases the symptoms subsided spontaneously, and in seven relief followed secondary operations. Deducting these 22 leaves in the last analysis 18 of the 99 cases with a return of symptoms, 18.1 per cent.

Classified according to the presence or absence of calculi one finds:

Calculous 69, with 33 return of symptoms (47.8 per cent.).
Cholecystostomies 45, with 25 return of symptoms (55.5 per cent.).
Cholecystectomies 24, with 8 return of symptoms(33⅓ per cent.).
Non-calculous 30, with 7 return of symptoms (23.3 per cent.).
Cholecystostomies 22, with 5 return of symptoms (22.7 per cent.).
Cholecystectomies 8, with 2 return of symptoms (25 per cent.).

In the calculous cases the return of symptoms was 47.8 per cent., twice as high as in the non-calculous cases, 23.3 per cent.; this can probably be explained by the fact that the non-calculous case is frequently less complicated or perhaps an earlier phase of calculous disease, therefore more amenable to permanent cure after suitable treatment. Of course the probability of mistaken diagnosis is much greater in the non-calculous than in the calculous type of the disease, if such errors could be excluded it would tend toward making the return of non-calculous cases smaller and emphasize the difference still more strongly.

Considered from the point of view of the type of operation one finds:

Cholecystostomies 67, with 30 return of symptoms (44.7 per cent.).
Calculous cholecystostomies 45, with 25 return of symptoms (55.5 per cent.).
Non-calculous cholecystostomies 22, with 5 return of symptoms (22.7 per cent.).
Cholecystectomies 32, with 10 return of symptoms (31.2 per cent.)
Calculous cholecystectomies 24, with 8 return of symptoms (33⅓ per cent.).
Non-calculous cholecystectomies 8, with 2 return of symptoms (25 per cent.).

The 13 per cent. higher return of symptoms following cholecystostomy as compared to cholecystectomy may be due to the fact that cholecystostomy (during these years 1910–1920) was more often performed in the advanced complicated and usually calculous cases, the return being twice as frequent in calculous as non-calculous cases. While after cholecystectomy, in presumably less complicated or earlier cases, the difference between the two types of disease is much less, only 8 per cent.

Of the 40 cases known to have return of symptoms, in 15 the symptoms subsided spontaneously, and 7 were relieved after secondary operation.

In a consideration of spontaneous subsidence of symptoms one must realize that in *recent* cases a remission of symptoms would be of little importance—as it is a well-known fact that symptoms of biliary tract disease are often absent or misinterpreted. Cases with known gall-stones often defy diagnosis between attacks of colic, though a properly developed history will usually bring out a story of "dyspepsia", "indigestion", "gas" with qualitative food distress. Long intervals between attacks of symptoms are not rare and are often attributed to the administration of some medicine or operation.

In these 15 cases of spontaneous subsidence of symptoms, the gall-bladder remains in 11 and has been removed in 4; the time of return of symptoms after operation was, first year in 11; fifth year in 1; sixth year in 2; seventh year in 1. The duration of symptoms was one year in 8; two years in 4; and three years in 3.

Of the 14 secondary operations death followed in 3, relief of symptoms was secured in 7, and symptoms persisted in 4.

The deaths were in the following cases:

1. Previous operation, cholecystostomy for acute calculous cholecystitis and cholangitis; at second operation, six weeks later, for continuation of same symptoms and increasing jaundice (thought to be due to stone in common duct), the common duct was opened and drained, no calculi nor obstruction was discovered.

2. Previous operation, cholecystostomy, for chronic calculous cholecystitis, followed by satisfactory recovery; but two years later a mass developed at the site of the operation; secondary operation revealed carcinoma, primary of the gall-bladder, at the site of previous cholecystostomy.

3. Previous operation, cholecystostomy, for chronic calculous cholecystitis and cardiospasm, which was followed by relief of symptoms for one and a half years and then by a recurrence of gastric symptoms. Secondary operation was gastrostomy for carcinoma of cardia. (The relationship between cholelithiasis and cardiospasm is not uncommon.)

Relief of symptoms followed secondary operations in seven cases.

1. Primary operation, cholecystostomy, non-calculous. Second operation, gastro-enterostomy for pyloric obstruction due to adhesions.

2. Primary operation, cholecystostomy, calculous. Second operation, drainage of pancreatic cyst.

3. Primary operation, cholecystostomy, non-calculous. Second operation, cholecystectomy, non-calculous.

4. Primary operation, cholecystostomy, non-calculous. Second operation, cholecystectomy, non-calculous.

5. Primary operation, cholecystostomy, calculous. Second operation, cholecystectomy, calculous.

6. Primary operation, cholecystostomy, calculous. Second operation, cholecystectomy, calculous.

7. Primary operation, cholecystectomy, calculous. Second operation, drainage of common duct, non-calculous, no obstruction.

The symptoms persisted after second operation in four cases.

1. Previous operation, cholecystostomy, calculous. Secondary operation, cholecystectomy, calculous.

2. Previous operation, cholecystostomy, calculous. Second operation, cholecystectomy, calculous.

3. Previous operation, cholecystostomy, calculous. Second operation, cholecystectomy, non-calculous, and drainage of common duct, non-calculous.

4. Previous operation, cholecystostomy, non-calculous. Second operation, cholecystostomy, non-calculous.

A review of the 14 secondary operations shows:

Primary operation was calculous in 12 and non-calculous in 2.

Primary operation was calculous and secondary operation non-calculous in 8: 1 common duct, death; 2 common duct, relief; 1 primary cancer of gall-bladder, death; 1 cancer of stomach, cardia, death; 1 pancreatic cyst, drain relief; 2 cholecystectomies, relief.

Primary operation was calculous and secondary operation calculous in 4 cholecystectomies, 2 relief and 2 no relief.

Primary operation was non-calculous and secondary non-calculous in 2: 1 gastro-enterostomy and cholecystostomy and 1 cholecystostomy repeated, 1 relief and 1 no relief.

Primary operation was non-calculous in 2, and secondary calculous in none.

Gall-bladder remains in 1 of 4 that were not relieved by second operation.

Gall-bladder remains in 2 of 7 that were relieved by second operation.

The 8 cases in which gall-stones were present at the primary operation and were absent at the secondary operation, in conjunction with numerous cases of this finding since 1920, calls attention to non-calculous intermittent biliary obstruction following cholecystectomy and the possible and probable influence of pancreatitis, hepatitis, duodenal ulcer or duodenitis and retro-peritoneal changes, as a cause of a return of symptoms.

Consideration of the 15 cases with persisting symptoms shows that the gall-bladder remains in 7 and has been removed in 8.

In 12 there were calculi, 6 cholecystostomies, and 6 cholecystectomies.

In 3 there were no calculi, 1 cholecystectomy and 2 cholecystostomies.

The symptoms complained of were "dyspepsia, indigestion," without colics in 4; with colics in 11.

The designation "neurotic" and "enteroptotic" was applied to 4.

Return of symptoms coincided with pregnancy in 1, cholecystostomy.

In 2 cases of cholecystectomy for calculi, Wassermann was 4 plus.

The appendix had been removed in all of this group of cases and in 7 cases pelvic operations had also been performed.

There were 15 females of the following ages: 29–30, 1; 30–40, 4; 40–50, 5; 50–60, 4; 60–70, 1.

Definite conclusions may not be drawn from such a review of so small a number of cases, the possible errors are too numerous, for example: In the calculous cases there is always the possibility of overlooked stones and of coincident disease, while in the non-calculous cases the chance of mistaken diagnosis is greater and the existence of intercurrent disease is, of course, the same.

Having demonstrated that unsatisfactory results, *i.e.*, return of symptoms follow gall-bladder operation, drainage or removal for calculous or non-calculous cholecystitis, the next question is what is the cause?

Among the many causes for return of symptoms one may mention: (1) Mistaken diagnosis; (2) surgical accidents; (3) incomplete operation; (4) coincident or resultant disease.

1. Mistaken diagnoses: Gall-bladder disease may be similated by a multitude of conditions, among the more common are the following: (a) Diseased appendix, not removed; (b) gastro-intestinal ulcer or malignancy; (c) Renal calculi or disease; (d) cardiac disease; (e) pulmonary disease; (f) lues or other nervous disease.

(a) A diseased appendix is rarely overlooked or allowed to remain as a source of symptoms after gall-bladder operation; in this day of aggressive surgery one might say that even a normal appendix is rarely overlooked or allowed to remain.

(b) Duodenal or gastric ulcers, small ones, on the posterior wall, or mucous erosions may certainly be overlooked, such ulcerations or malignancies are often coincident with biliary tract disease.

One must not be lured into a sense of false security by the gratifying discovery of gall-stones that confirms a pre-operative diagnosis. The adjacent viscera must be carefully examined as the coincidence of gall-stones with other disease is not uncommon. For example, in 1922 I made a diagnosis of gastric ulcer in a doctor of fifty-four years of age; he was soon operated upon, gall-stones were removed which was followed by a return of symptoms and in 1927 the patient died of cancer of the stomach.

(c), (d), and (e). The *acute* colic referred to the abdomen from the heart, angina pectoris; lung, pneumonia; kidney, renal colic; is a well-recognized clinical phenomenon; but it is not as well recognized that *chronic* disease of these extra-abdominal viscera may likewise cause chronic or sub-acute pain or distress in the upper abdomen which may be misinterpreted as biliary tract disease.

(f) Gastric crises of tabes is often overlooked as a cause of abdominal pain. In two of this series, gastric crises caused symptoms to persist after removal of calculi and gall-bladder.

Intercostal neuralgia has been recognized as a simulator of gall-bladder disease.

A frequent mistake is that in which the gall-bladder is normal, but is drained or removed under the impression that it is causing the symptoms.

The teaching that "gas on the stomach" with qualitative food intolerance justifies the diagnosis of cholecystitis and cholecystectomy as treatment, wonderfully simplifies the practice of medicine, but calls for careful scrutiny of *end* results, before it can be accepted.

Under the heading of mistaken diagnoses, one must consider the question, what constitutes a normal gall-bladder and how is it to be recognized? Quite a question when one realizes that it has not been answered entirely satisfactorily in connection with as accessible and visible a structure as the tonsil.

A gall-bladder may be abnormal and produce no clinical symptoms and per contra, the symptoms of cholecystitis may exist with a normal gall-bladder.

A gall-bladder may be normal to palpation and inspection, but show pathology after it is opened; and the gall-bladder that seems normal to the naked eye may prove to be pathologic, under the microscope.

Sterile bile does not necessarily mean normal gall-bladder wall.

So it would seem in this matter of correct diagnosis that one is "between the devil and the deep blue sea". If one operates upon clinical symptoms only, with usual signs of pathology absent, one is in danger of performing an unnecessary, therefore harmful operation, *i.e.*, removal of a normal gall-bladder. Or if one awaits the development of positive gross evidence of pathology in or around the gall-bladder, there is danger of intercurrent or resultant pathology in neighboring viscera having become established, with a consequent diminished chance of permanent cure.

(2) Surgical accidents: Damage to liver, pancreas, duodenum or common duct are uncommon, but injury to common duct is perhaps more frequently undetected at time of operation.

(3) Incomplete operation, stone remaining in ducts or portion of gall-bladder or cystic duct remaining, may of course cause a continuation or recurrence of symptoms.

Overlooked calculi or recurrence of calculi does not seem to be responsible for a large number of recurrences. For instance, Judd and Burden failed to find calculi in 24 secondary operations upon the common duct. In this series the common duct was explored in 3 of the secondary operations and no calculi were found. In 12 cases of primary operation for calculi, at 8 secondary operations no calculi were found.

It would seem reasonable to conclude that the most usual cause of return or continuation of symptoms *after* operation are the same factors that frequently cause a return or continuation of symptoms *before* operation, namely disease in viscera other than the gall-bladder.

(4) Coincident or resultant disease:

Extension of disease from the gall-bladder and the extrahepatic ducts to the intrahepatic ducts and the liver, with perhaps, biliary cirrhosis, is a logical sequence. Cirrhosis of the portal type may be coincidental. Acute

pancreatitis is often an extension of biliary tract disease, and the chronic type has been demonstrated as a sequence and may cause persistent symptoms. In this series the pancreas was diseased in 42 cases (22.5 per cent.) in 4 carcinomas; in 9 acute pancreatitis; in 29 chronic pancreatitis, interstitial or lymphatic. Gastritis as evidenced by hypo- or achlorhydria is a recognized sequence of gall-bladder disease. So-called colitis is often a coincidence, if not a causative or resultant factor in biliary tract disease. The relationship between cholecystitis and myocarditis has been suspected as being a causal one. Gastric or duodenal ulcer or appendicitis are not infrequently coincidental with possible causative relationships. An infected gall-bladder may act as a focus of infection with the usual secondary manifestations.

Post-operative adhesions are, of course, always present, but in rare instances they may cause crippling deformities that interfere with function in neighboring hollow viscera and thereby cause post-operative symptoms.

In order to reduce the percentage of unsatisfactory results it must be recognized that surgery is a means to an end and not an end in itself. Cholecystostomy, cholecystectomy or choledochotomy may save life, but in many cases gall-stones are merely an incident in the course of cholecystitis and cholecystitis is often merely an incident in a biliary tract disease, that calls for medical management before and after drainage or removal of the gall-bladder; once again demonstrating the need of coöperation between the physician and the surgeon; and that the surgeon, unless he degenerates into a mere technician, must be a physician who operates.

NON-PARASITIC CYSTS OF THE LIVER[1]

By Alexius McGlannan, M.D.

of Baltimore, Md.

Cysts are an interesting group of tumors and a study of their origin opens an attractive field for speculation. One group of cysts is formed by the persistence of fetal structures. Inclusion of embryonic or adult cell groups produces certain types. Occlusion of ducts by faulty development or by fetal or extrauterine neoplastic or inflammatory processes explains the origin of others. Hemorrhage, especially that into parenchymatous organs or vascular tumors may result in cyst formation during the organization of the clot. Obstruction of lymphatic channels or spaces leads to their cystic dilatation. Degeneration involving the tissues or the structure of solid tumors is responsible for cystic changes in various parts of the body. Certain types of neoplastic growths are cystic in their ordinary development.

Parasitic cysts are special forms of reaction to invasion by certain organisms. The late Doctor Thompson in an address before the Southern Surgical Association in 1919 showed how cysts developing from embryonic inclusions or remnants may be carried far from the seat of their origin by the growth of the normal structures.

Non-parasitic cysts of the liver may be single or multiple. Multiple cysts may be limited to the liver, or may be associated with cysts of the kidneys, the pancreas or other abdominal organs. In many cases patients suffering from this polycystic disease show external evidences of defects in development, such as cleft palate, polydactylism, meningocele, etc.

The single cysts appear to be developed from aberrant bile ducts or to be cystic adenomata developed from the same source. The wall of these cysts is lined either entirely or in part by epithelium of the same type as that lining the normal bile ducts modified by the size of the cysts.

Ordinarily there is no evidence of the formation of secreting epithelium. As a rule the cysts do not communicate with the biliary tract, and the contents rarely contain bile. Of sixty-three cases studied by J. F. X. Jones,[2] only four are reported to have contained fluid resembling bile. In one of these four the operator's diagnosis was congenital dilation of the gall-bladder and of the common duct.

If these cysts are primarily cystic adenomata, secreting epithelium of an embryonic type at some time formed part of their structure. The duct channels being imperfectly developed and closed, an accumulation of secretion takes place within the confines of the tumor, producing the cyst.

[1] Read before the Southern Surgical Association at Augusta, Ga., 1927.

[2] Annals of Surgery, January, 1923, vol. lxxvii, p. 68.

The secretion of imperfect cells is not likely to be true bile. It accumulates under varying degrees of tension, and is subject to chemical changes during the period of accumulation and retention within the cyst cavity. Such physical and chemical action has a marked influence on the structure of the cyst wall, on the nature of its contents, and on the relations of the cavity to the adjacent normal liver. When a flow of bile occurred during the course of healing after marsupialization and drainage, as in the case reported by Maes,[3] the cysts all seem to have been large central ones, where the probability of rupture of a duct into the cavity could explain the flow of bile into the drainage tract. The changes mentioned above show the impracticability of determining the exact origin of these large solitary cysts by a study of the structure of the wall or the chemical composition of the contents.

Multiple cysts of the liver may be the result of similar occlusion of aberrant bile ducts. The diffuse involvement of the liver makes it unlikely that they are cystic adenomata. The association of multiple cysts of the pancreas may be explained by their similarity in origin, both organs developing from buds on the embryonic duodenum.

It is more difficult to explain the frequent association of congenital polycystic disease of the kidney with multiple cysts of the liver. The most generally accepted theory regarding the cause of the kidney disease attributes it to a developmental error in the fusion of the embryonic kidney, which leaves certain glomeruli and secreting tubules ununited to the corresponding excretory tubules. As the secretion collects in these occluded areas, cysts of various size and shape are formed.

Except for the fact that multiple congenital defects are common, there is no reasonable explanation for the simultaneous occurrence of such a mishap in the embryonic kidney and the development of aberrant bile ducts in the liver.

Carling and Hicks[4] believe that these polycystic lesions are formed from inclusion of isolated portions of the Wolffian body in the enteric buds forming the liver and pancreas and in that part of the intermediate cell mass in which the secreting portion of the kidney is developed.

Most solitary cysts are found growing from the under surface of the liver, extending to the anterior border in the region of the gall-bladder fossa. Teuscher[5] states that the developmental anomaly producing liver cysts involves more especially the embryonic plexus like hepatic ducts at the hilum.

A number of cases are reported in which the cyst occurred in the central areas of the liver, and presented on either the superior or inferior surface. Several cysts appeared as pedunculated tumors growing from the surface or margin of the liver. The central cysts usually are surrounded by a zone of fibrosed liver tissue. Atypical lobulation of the liver may be the result of pressure of a large cyst or may be an evidence of congenital malformation.

The condition is more common in females, the proportion of incidence

[3] American Journal of Surgery, March, 1924, vol. xxxviii, p. 68.

[4] British Journal of Surgery, October, 1925, vol. xiii, p. 238.

[5] Archives of Pathology and Laboratory Medicine, April, 1927, vol. iii, p. 706.

between the sexes being about 4 to 1. Most of the patients are between forty and sixty years of age, although there are cases reported occurring in newborn children and in septuagenarians. External violence is mentioned in a few cases as a possible cause, but no proof is offered to show that trauma is a factor in the production of the cyst.

Clinically, solitary non-parasitic cysts of the liver have no signs or symptoms sufficiently characteristic to make a pre-operative diagnosis certain. They have been found when the abdomen was opened for exploration, or for the relief of symptoms interpreted as chronic cholecystitis, hydronephrosis, mesenteric cyst, ovarian cyst, tuberculous peritonitis and other lesions. When the diagnosis has been made as in the three cases from the Mayo Clinic included in Jones' report, they were large cysts recognized as tumors growing from the liver.

Many of these cysts grow in the neighborhood of the gall-bladder fossa and give rise to pressure on the gall-bladder or the extra-hepatic ducts. This pressure may explain the jaundice occasionally met with in connection with the cysts. It is probably responsible for the symptoms like those of cholecystitis which are so often noted, and may be the cause of the coincident evidences of chronic inflammation found in the gall-bladder and ducts.

Examination by means of the X-ray may show the relation of the abdominal tumor to the liver, and thereby aid in the differentiation from ovarian and other cysts. Calcareous deposits are more common in parasitic cysts, but Sharkey[6] reports such areas in the thin wall of a solitary cyst. The shadow of such spots might help in this differentiation. Pneumoperitoneum should make this examination more accurate in its results.

When the physical examination and the history suggest a solitary cyst of the liver, further investigation should be made regarding the probability of echinococcus infection as its cause.

At operation it is necessary to distinguish the simple cyst from the parasitic one, and from cystic dilatation or diverticulum of the extra-hepatic bile ducts. The last-mentioned lesion is recognized by its anatomical relations and is treated by making an anastomosis between the dilated duct and the gastro-intestinal tract. The typical hydatid cyst has a laminated outer capsule or adventitia and an inner membrane which carries the daughter cysts, while the simple cyst has a thin single wall.

Solitary cysts are treated by excision, complete or partial, or by drainage with or without marsupialization.

The following is an abstract of the record of a personal case of solitary non-parasitic cyst of the liver.

Case Report.—S–47974. The patient, a white matron, age thirty, was admitted to the Mercy Hospital, May 10, 1926, with a diagnosis of chronic cholecystitis. The diagnosis was made by her physician, Dr. Wilbur Pearce, and I concurred in its correctness. For more than a year she had suffered from recurrent attacks of pain in the upper right quadrant of the abdomen. The pain radiated to the back and to the right shoulder. With

[6] Trans. London Patholog. Soc., 1882, vol. xxxiii, p. 168.

each attack there was vomiting, and more or less indigestion was present between them. There was no history of jaundice. Since the last attack, ten days before admission, an indefinite but apparently smooth rounded mass could be felt at the margin of the liver and was taken to be a distended gall-bladder. This area was tender and there was some muscle spasm present.

At operation we found a cystic tumor growing from the lower surface of the liver and extending a short distance beyond its anterior margin. The tumor was thin-walled, lobulated and sausage-shaped, about 9 inches in its long and 2½ in its transverse diameter. The omentum was adherent to it at several points. The cyst was situated just to the left of the gall-bladder, but was not attached to this organ or to its ducts. There were no evidences of disease of the bile tract.

Aspiration drew out about 700 c.c. of clear straw-colored fluid which did not stain the gauze packs. After the cyst was empty we found that part of the wall was so intimately blended with the liver substance that no line of separation could be discovered. For this reason the greater portion of the wall was excised, leaving a sufficient amount to suture into a narrow tube, the inner side of which included the attachment to the liver. This tube was packed with iodoform gauze and brought up to, but not sutured to the peritoneum. The abdominal wall was closed around the protruding pack.

The patient recovered without any complications. A small amount of fluid which scarcely stained the dressings drained out for the first week. The iodoform pack was gradually removed, the last part coming out on the tenth day. The wound then healed and the patient was discharged from the hospital twenty-seven days after the operation. She has had no recurrence of abdominal symptoms and is considered cured.

Section of the cyst wall shows fibrous tissue containing many large new blood-vessels, a certain number of bile ducts and an area of liver cells which are entirely surrounded by fibrous tissue and do not appear to connect with any of the ducts.

PHLEGMONOUS GASTRITIS

OF BACILLUS ÆROGENES CAPSULATUS (B. WELCHII) ORIGIN

By John J. Morton, M.D.

AND

Samuel J. Stabins, M.D.

of Rochester, N. Y.

FROM THE DEPARTMENT OF SURGERY, UNIVERSITY OF ROCHESTER SCHOOL OF MEDICINE AND DENTISTRY

Phlegmonous gastritis is in itself of sufficient rarity to always excite comment. The occurrence of the bacillus ærogenes capsulatus as the probable etiologic agent or at least as the most prominent factor in the picture has led us to report the following observation.

Case Report.—A rather thin, sallow looking but otherwise healthy man, seventy-two years, had been running an elevator until two days ago, October 23, 1927. At that time he noticed slight discomfort in the stomach but attributed this to overeating. The trouble persisted, however, and at 11 A.M. on October 25 while working was seized with a sudden, severe but not sharply localized pain in the upper part of the abdomen. He was obliged to stop work and was admitted to the Emergency Division. There was no vomiting, although slight nausea was present. On examination, the abdomen was soft. The bowels had moved normally during the morning before the pain came on. He was not considered to be in serious condition and was not seen again till 3 P.M. when he was admitted to the medical service for further study. At 4 P.M. it was apparent that the patient was suffering with a very severe pain in the epigastrium. His temperature was 37.8° C., the pulse rate was 92 and the respirations were 20. General examination showed moderate arteriosclerosis; a few râles at the base of the left lung, a blood pressure of 165 systolic and 100 diastolic; and a normal heart. The abdomen showed the scars of two hernia operations with a slight defect still present on the right. There was acute tenderness over the entire abdomen and a board-like rigidity. The dulness of the liver was at the costal margin at the start of the examination but fifteen minutes later there was tympany replacing the dull area. The count of the white blood-cells showed 17,300. The only point of importance in his past history was two attacks of previous abdominal discomfort never accurately diagnosed. In one of these attacks he was in bed six weeks.

A diagnosis of acute perforated ulcer was made. Exploratory operation was carried out under nitrous oxide anæsthesia. Upon opening the peritoneum a fluid with greenish undigested food particles was encountered and a few bubbles of gas escaped. A perforation 1 cm. in diameter was found on the anterior surface of the duodenum just beyond the pylorus. A few recent adhesions were also present. A non-indurated ulcer was excised by the Heineke-Mikulicz procedure and closure was made without drainage. The patient did not react well to his anæsthesia and was in poor condition on leaving the operating room. His pulse was rapid and feeble; his blood pressure had dropped to 100 systolic and 80 diastolic, and was fluctuating; and he was very pallid. He rallied under hypodermoclysis and rectal fluids and was in fairly good condition the next morning. Culture of the fluid obtained from the peritoneum at operation showed no growth.

He continued to improve slowly for five days till November 1, 1927, although he differed from the average perforated ulcer patient in repeatedly vomiting small amounts of "coffee ground" material and in running a pulse rate at 120 per minute. On November

1, 1927, an area of relative dulness was found at the base of the left lung posteriorly extending upward to the angle of the scapula. The breath sounds were unchanged in this area but there were fine crackling râles present. Röntgen-ray pictures suggested consolidation in the left lower lung field with the question of fluid as well. The abdomen was considerably distended, tympanitic throughout and held fairly rigidly. The white blood count was 18,500. He was irrational through the day. The temperature rose to 37.6° C.; whereas it had been normal or subnormal up to this time, and his respirations were rapid and shallow at 34 per minute.

A Levene tube was introduced into the stomach on account of the vomiting and 1000 c.c. of dark brown fluid recovered. An additional 2000 c.c. drained into the bottle from this tube during the night. The material did not have a fecal odor. The following morning the vital signs showed little change except that the pulse was feeble and obtained at the wrist with difficulty. He was evidently in extreme distress due to embarrassment in respiratory exchange and incessant retching. He gradually grew worse during the morning: the blood pressure could not be recorded; he became pulseless; the skin over the entire body was cold, ashy gray and clammy. He remained conscious though irrational for two hours after this marked picture of shock had appeared. Then he vomited suddenly nearly 1000 c.c. of bright red fluid and died immediately thereafter.

FIG. 1.—Photograph taken from colored drawing. It shows thickened and œdematous mucosa studded with gas bubbles.

At post-mortem examination within one hour of his death, the peritoneal cavity contained about 500 c.c. of clear serous fluid of yellowish tinge. There were old adhesions about the appendix region and fresh easily separated adhesions about the transverse colon, stomach and gall-bladder. Each pleural cavity contained about 2000 c.c. of straw-colored fluid. The heart was normal except for myocardial scarring and advanced sclerosis. The lungs showed hypostatic congestion and œdema but there were no patches of pneumonia. The pyloric end of the stomach was covered with omentum

which had been sutured in place. On stripping away the omentum a line of silk sutures with no obvious inflammation about them was exposed. The stomach did not present anything unusual from its external surface. The mucosa of the lower 10 cm. of the œsophagus was boggy, blood stained, and acutely inflamed. The stomach contained considerable brick-colored blood mixed with mucus and food material. The mucosa was thickened and œdematous; it was dark reddish-brown in color; it contained bright red extravasated blood and was studded with gas bubbles about the size of a pin head. The rugæ were prominent on account of the infiltration with inflammatory exudate. The mucosa underlying the sutures was gray and necrotic over an area of 2 cm. No definite source of hemorrhage was found. Cultures were made from the underlying tissue. The stomach wall appeared to be about 1 cm. in thickness in contrast to the average. The entire stomach, lower 10 cm. of the œsophagus and upper 10 cm. of the duodenum was involved in the picture but no gas bubbles were present in the œsophagus or duodenum. (Fig. 1.) The remainder of the gastro-intestinal tract was negative. There was no evidence of gas bacillus infection in any other portion of the body, indicating that this was a localized process and not part of a general terminal gas bacillus invasion. Aside from arteriosclerosis nothing else of importance was noted.

Microscopic picture through the fundus of the stomach showed the mucosa diffusely infiltrated with mono and polymorphonuclear cells, the former predominating. The submucosa was approximately five times its usual thickness. There were occasional polymorphonuclear and lymphoid cells in both the submucosa and muscularis. The gas bubbles were present in the mucosa, muscularis mucosa and submucosa especially, but not in the muscularis or serosa. (Figs. 2 and 3.) Numerous bacteria were also noted in the submucosa, the predominating organism being a large, blunt, Gram-positive bacillus.

Sections through the operative area at the pylorus showed that part of the mucosa was necrotic. The submucosa was not as greatly distended as in the fundus but contained many more pus cells. The muscularis was also acutely inflamed and partially destroyed by abscess formation. Both sections included thrombosed veins. The microscopic picture of gas bacillus infection was not found in any other organ.

Bacteriology.—Direct smears taken at post-mortem from the stomach wall after cutting through the mucosa showed numerous Gram-positive bacilli and in places chains of cocci. (Figs. 4 and 5.) Anaërobic culture in deep litmus milk showed acid reaction and stormy fermentation characteristic of B. Welchii. In deep meat broth there was a turbid growth with considerable gas production. The Bacteriological Department considered that further tests were not necessary to establish the identity of the suspected organism so that serologic examination and animal inoculation were not carried out.

This man at first did not seem to react differently from the usual post-operative perforated ulcer patient. Recovery was not as rapid or as marked but it was thought that his age and general condition were responsible. Small quantities of "coffee ground" material were vomited daily post-operatively so that hemorrhage from one of the gastric vessels was suspected. Clinical evidence of a severe blood loss was not obtained, however, and expectant treatment was accordingly adopted. The picture changed decidedly for the worse five days after the onset but enough signs were found in the chest to account for it. At this time the condition was such as one finds in a severe toxæmia, and almost identical in retrospect with the rather common war toxæmia associated with gas gangrene. The accumulation of fluid in the stomach indicated a disturbed motor or secretory function of this organ but the character of the vomitus gave no evidence of a bowel obstruction below. The invasion of the stomach by the Welch bacillus was not suspected clin-

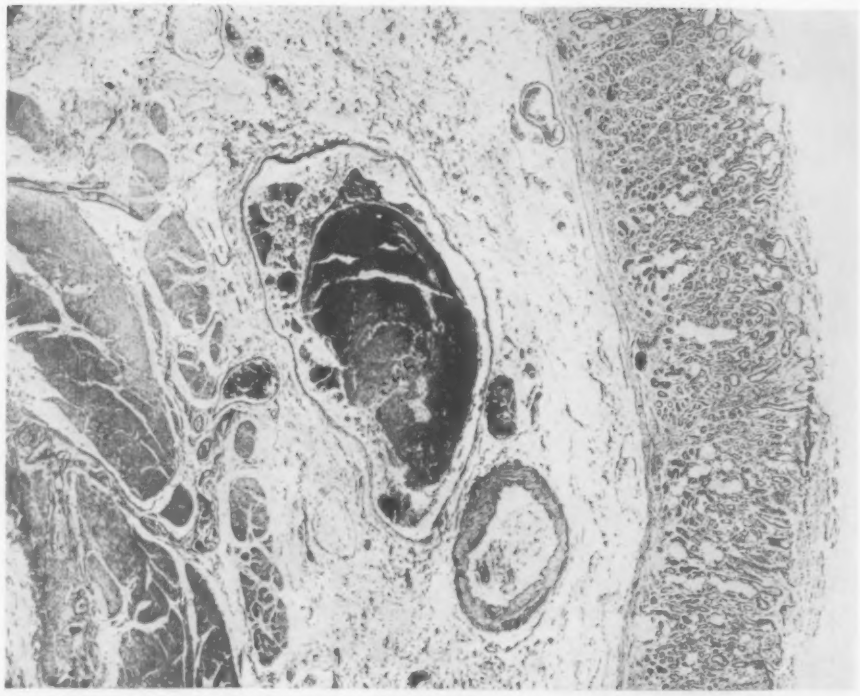


FIG. 2.—Microphotograph of section through the wall of the stomach. (x 10.) The gas bubbles show very plainly in the mucosa, muscularis mucosa and less well in the submucous layer through lack of contrast. The muscular layers are not involved. A large thrombosed vein is present in the centre of the microphotograph.

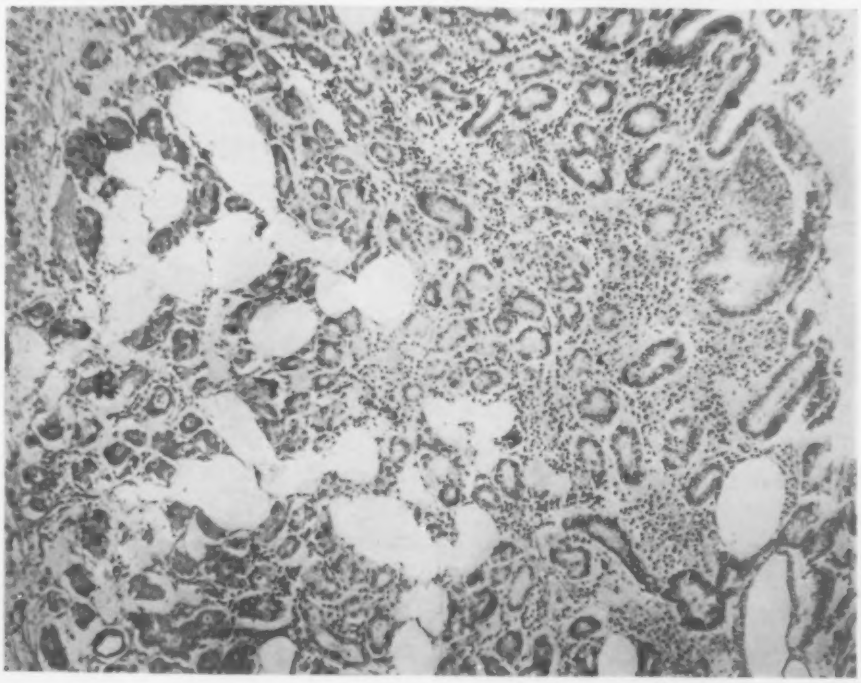


FIG. 3.—Higher magnification. (x 96.) The section consists mainly of mucosa with contained air bubbles.

ically although it probably occurred at least from twenty-four to thirty-six hours before death and perhaps even earlier. The probable portal of entry was in the necrotic area about the suture line. The suture line itself was intact with no evidence of leakage or peritonitis.

There is quite an extensive literature dealing with phlegmonous gastritis. Lawrence,[1] who has reviewed the subject thoroughly, says that up to the time of his report there were but two hundred and thirty-seven cases recorded. He added two other case reports. In summarizing the etiology, he found the streptococcus group to be the most common causative agents; occasionally also staphylococci and rarely pneumococci were responsible. Sometimes one of these organisms occurred in a mixed infection with B. coli and B. subtilis. In no instance, as far as we are aware, has the B. ærogenes capsulatus played a rôle either as the primary cause or as a secondary invader.

It seems rather strange, in view of the prevalence of the Welch bacillus as an inhabitant of the big bowel of man, that infection with this organism takes place so rarely. In the presence of trauma, ulcerations, surgical interventions, etc., ample opportunity would be provided for invasion with this bacillus. Apparently usually the local conditions are not favorable for the lodgement or propagation of this organism even in the large intestine. Normally the lower part of the small intestine contains a very active bacterial flora, whereas the stomach and upper intestine are relatively sterile. This was shown by Cushing and Livingood[2] in a careful study. It has only recently been demonstrated that under altered physiological or pathological conditions, invasion of the whole gastro-intestinal tract is possible (Meleney, Jobling and Berg[3]; MacNeal and Chace[4]; Gorke[5]). We have personally recovered B. Welchii repeatedly from high jejunostomy loops in patients suffering from obstruction or ileus due to any cause.

The gastro-intestinal tract, however, is not entirely immune to the invasion and attack of the B. Welchii. Deutsch[6] reported a case of gas gangrene of the right arm with the portal of entry through the oral mucosa about an ulcerated tooth. The organism has also been recorded in various parts of the body (Greeley[7]), who reports two cases of cholecystitis due to B. Welchii. Gould and Whitby[8] likewise reported a case of cholecystitis due to this organism. Mix[9] presented a very complicated picture of a persistent fecal fistula following appendectomy. He believed it to be due to B. Welchii. It was successfully treated by withdrawal of carbohydrates.

Typhoid fever has been complicated by Welch bacillus infection in nine instances according to Muller and Lincoln.[10] In one group there was no perforation but a general B. Welchii infection by invasion through the ulcers; in a second group there was a perforation of the ulcer and gas bacillus in the peritoneal exudate but no visceral involvement. Case VIII (Howard) comes the closest to duplicating ours because a gastric ulcer served as the probable portal of entry.

In the pathological picture of phlegmonous gastritis the process appears to be chiefly limited to the submucosa. In our case, the gas bubbles indicate

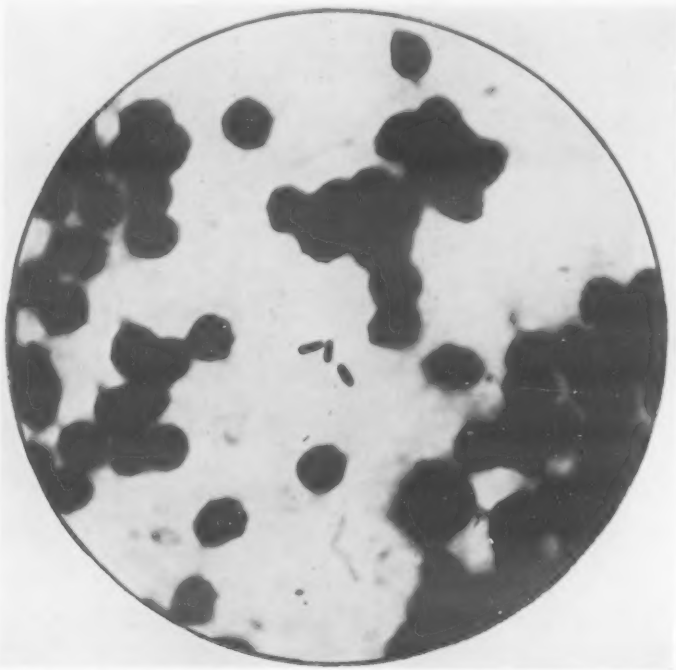


FIG. 4.—The characteristic blunt Gram-positive short rods of the B. Welchii present in a stained smear preparation made from the submucous layer. (x 56.)

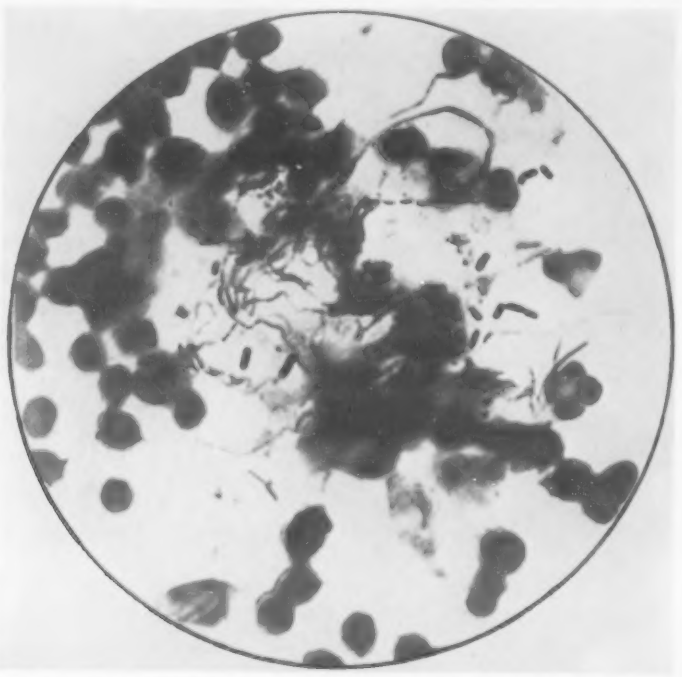


FIG. 5.—Chains of streptococci and other organisms as well as abundance of Bacillus Welchii rods from a stained smear preparation made near the suture line. (x 56.)

a marked submucosal involvement but the mucosa also shows much damage. The muscular coats which one would presume to be especially susceptible, seem to have escaped entirely.

The gastric tissues show large numbers of bacilli with the morphological characteristics of B. Welchii. In the region of the gangrenous patch there were numerous chains of streptococci as well. We believe that these organisms represent the secondary invaders in this case and that the predominant element in the production of this phlegmonous gastritis was the Welch bacillus.

BIBLIOGRAPHY

[1] Lawrence, J.: Phlegmonous Gastritis. Boston M. & S. J., vol. cxcv, p. 800, 1926.

[2] Cushing, Harvey, and Livingood, Louis E.: Experimental Study upon the Bacteriology of the Upper Portion of the Alimentary Canal. Johns Hopkins Hosp. Rep., vol. ix, p. 543, 1900.

[3] Meleney, F. L., Jobling, J. W., and Berg, B. N.: Experimental Chronic Duodenal Obstruction. Arch. Surg., vol. xiv, p. 752, 1927.

[4] MacNeal, N. J., and Chace, A. F.: A Contribution to the Bacteriology of the Duodenum. Arch. Int. Med., vol. xii, p. 178, 1913.

[5] Gorke, H.: Ueber die Bakteriologie des Duodenalsaftes Mitt. a. d. Grenzgeb d. Med. u. Chir., vol. xxxv, p. 279, 1922.

[6] Deutsch, J. A.: Peculiar Case of Gas Gangrene. Med. Klin., vol. xvi, p. 652, 1920.

[7] Greeley, H.: Idiopathic Bacillus Ærogenes Capsulatus Infection. Johns Hopkins Hosp. Bull., vol. xxix, p. 231, 1918.

[8] Gould, E. P., and Whitby, L. E. H.: A Case of Bacillus Welchii Cholecystitis. Brit. J. Surg., vol. xiv, p. 646, 1927.

[9] Mix, C. L.: Bacillus Ærogenes Capsulatus Infection of the Intestines. M. Clin. Chicago, vol. ii, p. 1007, 1916.

[10] Muller, H. R., and Lincoln, J. S.: Bacillus Ærogenes Capsulatus Infection Complicating Typhoid. M. Rec., vol. ci, p. 190, 1922.

LATE RESULTS IN PERFORATED GASTRO-DUODENAL ULCERS *

By Richard Lewisohn, M.D.
of New York, N. Y.

FROM THE SURGICAL SERVICE OF DR. A. A. BERG, MOUNT SINAI HOSPITAL

Many articles have been written during the last ten years on the subject of late results following surgical treatment of chronic gastro-duodenal ulcers. On the other hand comparatively few papers have been published on the late results in perforated gastro-duodenal ulcers.

Eliot published in 1912 a very thorough study of immediate and late results following acute perforations of gastro-duodenal ulcers. However, most of the papers written on the subject of acute perforations confine themselves to a discussion of various surgical procedures and an investigation of the operative mortality.

It is stated very often as a positive fact that an acute perforation always leads to a complete healing of the ulcer with a formation of a simple scar. How this process of healing occurs does not seem to be quite clear to most authors. The acute perforation usually takes place in the centre of the ulcer. Although the perforation is only pin-point in size, the ulcer-area often varies from the size of a dime to that of a quarter. In the majority of cases the sutures which close the perforation have to be inserted in the area of acute inflammation. In spite of the fact that the inflamed area is not excised, most authors assume that a spontaneous process of complete healing follows an acute perforation.

One of the well-known procedures in the surgical treatment of chronic gastro-duodenal ulcers is based on the assumption that a spontaneous cure follows an acute perforation. Thus, the perforation is artificially produced by a cautery and the opening closed by sutures.

It seemed advisable to study the late results following acute perforation of gastro-duodenal ulcers with the same care which we used in the study of gastro-jejunal ulcers following gastro-enterostomy. The latter investigation brought to light the interesting fact that, at least in our material, gastro-jejunal ulcers occurred in 34 per cent. of the cases, with 18 per cent. proven by re-operation. The marked difference between this high figure and the usually accepted incidence of gastro-jejunal ulcers (about 5 per cent.) was explained by the fact that we studied our group very thoroughly in the follow-up clinic and did not rely on questionnaires, letters, etc.

In this paper I would like to present our conclusions as to permanent cures following the suture of acute perforated gastro-duodenal ulcers.

Stenbuck in a recent paper reviewed the operative causes of mortality following operations for perforated gastric and duodenal ulcers at Mount Sinai

* Read before the New York Surgical Society, February 22, 1928.

Hospital from 1915 to 1925. The mortality was 31 per cent. (twenty-seven deaths among eighty-eight cases).

With his kind permission I have used the same material with the object of getting some definite data as to end-results following operations for perforated gastro-duodenal ulcers.

In compiling these statistical data the same principles were employed as in the previous investigation on gastro-jejunal ulcers. Only those patients were included in this review who presented themselves personally in our follow-up clinic where we were able to study them carefully. As stated above, the only safe follow-up system for tabulating end-results is a thorough personal examination of the patient, with correlation of the X-ray findings and test-meals whenever these investigations are deemed important.

Thirty-three patients operated between 1915 and 1925 on the Surgical Services of Mount Sinai Hospital presented themselves in our return clinic for reëxamination. Years of operation are given in Table I.

TABLE I.

Distribution of Cases 1915–1925.

Year	Number of cases
1915	5
1916	4
1917	2
1918	2
1919	2
1920	3
1921	–
1922	4
1923	6
1924	3
1925	2

Twenty patients have been perfectly well and free from any gastric symptoms since the closure of the perforation. The perforation was located either at the pylorus or in the duodenum in eighteen cases. An exact differentiation between pyloric or duodenal ulcer is often impossible in acute perforations, as the inflammatory process makes the landmarks (for instance the pyloric vein) practically invisible.

In two cases the perforation was situated near the reëntrant angle.

In ten cases the operation consisted in simple closure of the perforation. In the other ten cases a gastro-enterostomy had been added, with or without pyloric exclusion (Berg's method). In six of these cases a Murphy button had been employed, which was used extensively on this service up to 1920.

Thorough X-ray examinations were performed in a considerable number of this group for two reasons: (1) We feel that in a follow-up clinic radiography ought to be employed freely, as this method gives us a very clear picture of the function of the stomach (size, emptying time, etc.), and (2) We were

interested to find out whether we could differentiate by an X-ray examination cases in which the ulcer had completely healed from those which were still suffering from a more or less active ulcer.

It seemed possible that the duodenum might show a perfectly normal bulb in those cases in which the ulcer had evidently healed completely. However, Doctor Goldfarb, who studied these groups for me, reports that a deformity of the bulb persists as a result of the layer suture of the perforation even in those cases which have been free from ulcer symptoms since the occurrence of the acute perforation. In other words, in the presence of a previous operation on the pylorus or duodenum a deformed bulb is not a definite indication for the presence of a recurrent ulcer. On the other hand marked tenderness of this region during the fluoroscopic examination, niches and retention, indicate that the patient still suffers from an inflammatory process at or near the pylorus.

I wish to discuss somewhat more in detail the group of failures, comprising thirteen cases. (Table II.)

Eight cases (No. 1, 2, 3, 6, 7, 8, 9, 10) are undoubtedly suffering from a recurrent ulcer. They have the seasonal attacks of epigastric pains, constipation, heartburn and sour eructations. While they have no retention in the stomach, they have to be very careful with their diet. A number of them have not been able to go back to work.

One patient (Case 10) has a pyloric stenosis. He passes the stomach tube every day and obtains a quart of fluid and semi-solid material. We advised re-operation (subtotal gastrectomy) to which he has not consented.

Another patient (Case 4) had three subsequent operations one and two years after the primary perforation. I feel sure that if I had performed a subtotal gastrectomy, when he reëntered the hospital the first time following the perforation, this patient would have been cured completely. These operations were performed, before we had instituted subtotal gastrectomy as a method of choice both for gastro-duodenal and gastro-jejunal ulcers.

Case 5 had a suture of the acute perforation with a gastro-enterostomy performed in 1920. He developed a gastro-jejunal ulcer and was operated by another surgeon. Re-operation for recurrent symptoms in 1924 revealed a duodenal ulcer perforated into the pancreas. A partial gastrectomy was performed. He made an uneventful recovery. He did not become anacid. He has the clinical and radiographic evidences of a gastro-jejunal ulcer. This patient belongs to the very rare group of a re-formation of an ulcer after resection of the stomach.

Two cases (11 and 13) in which a simple closure of the perforation had been performed one year previously, came to re-operation on account of the severity of recurrent symptoms. In the first case, two ulcers were found, one at the site of the old perforation and another (kissing ulcer) on the posterior wall of the duodenum. In the other case (No. 13) the perforated gastric ulcer had healed, but a penetrating duodenal ulcer was the cause of the persistent symptoms. Both patients were subjected to subtotal gastrectomy and made

TABLE II.

Follow-up Notes in 13 Cases of Perforated Gastro-duodenal Ulcers with Recurrent Symptoms.

Case	Year	Name	Location of ulcer. Operation	Subsequent course
1	1916	M.W.	Duodenal. Suture	1927: Epigastric distress. Sour eructations. X-ray examination shows recurrent ulcer.
2	1916	F.R.	Duodenal. Suture	1927: Sour eructations, pains. X-ray examination shows irregular bulb.
3	1917	J.T.	Duodenal. Suture. Gastro-enterostomy	1927: Pains, belching, blood in stool. X-ray examination shows: gastro-enterostomy normal; nothing going through pylorus. Diagnosis: recurrent ulcer.
4	1919	S.K.	Duodenal. Suture	1920: Re-perforation, walled off by liver. Suture of perforation, gastro-enterostomy. 1921: May: Gastro-jejunal ulcer. Disconnection of stoma. Ulcer excised. Jejunum closed. New gastro-enterostomy. 1921: December: Large recurrent gastro-jejunal ulcer. Jejunostomy. 1927: Patient still suffering. Does not want another operation (subtotal gastrectomy.)
5	1920	C.E.	Pyloric. Suture. Gastro-enterostomy	1921: Gastro-jejunal ulcer. Excision of ulcer. Disconnection of stoma. 1924: Partial gastrectomy for ulcer in posterior wall of duodenum with perforation into pancreas. 1927: Patient has clinical and radiographic evidences of recurrent gastro-jejunal ulcer.
6	1920	L.S.	Pyloric. Suture	1927: Patient felt well until 1922. Recurrent attacks once or twice a year. X-ray examination shows a niche and a constriction in the duodenum.
7	1922	A.R.	Pyloric. Suture	1927: Patient has a 27-year-old history of ulcer. Symptoms still persisting. X-ray examination shows an irregular bulb and 1/5 residue after 6 hours.
8	1923	M.D.	Pyloric. Suture	1927: 3 marked recurrences since operation, 2 in 1924, 1 in 1926. Patient is just recovering from 4th recurrence. X-ray examination shows a marked deformity of the duodenal bulb and hypermotility of the stomach.
9	1923	A.D.	Duodenal. Suture	1927: Sour eructations, pain, occasional vomiting. X-ray examination shows irregular bulb.
10	1923	M.L.	Duodenal. Suture	1927: Patient has typical symptoms of recurrent duodenal ulcer. Refused X-ray examination.
11	1924	E.F.	Duodenal. Suture	1925: Subtotal gastrectomy for recurrent duodenal ulcers. Findings: one ulcer at site of old perforation, another ulcer on posterior wall of duodenum, adherent to pancreas. 1927: Perfectly well.

TABLE II.—*Continued*

Follow-up Notes in 13 Cases of Perforated Gastro-duodenal Ulcers with Recurrent Symptoms.

Case	Year	Name	Location of ulcer. Operation	Subsequent course
12	1924	J.H.	Pyloric. Suture	1927: Patient felt well for 3 months. Since then symptoms have recurred. Marked hunger pain. Patient uses tube every day and drains about one quart from stomach. X-ray examination shows marked retention.
13	1925	P.K.	Gastric at reëntrant angle. Suture	1926: Subtotal gastrectomy for recurrent duodenal ulcer. Findings: scar of previously closed gastric ulcer. Ulcer at posterior wall of duodenum, perforated into pancreas. 1927: Perfectly well.

an uneventful recovery. They are perfectly well and free from any gastric symptoms.

I have recently re-operated a patient, who had been operated on this service for an acute perforation in 1926. This case is reported on page 955. He was not included in this statistical survey. Since we define late results as those occurring after at least two years, we carried our reëxaminations up to 1925. Re-operation of this patient revealed an active ulcer, perforated into the head of the pancreas.

A number of patients were subjected to a partial or subtotal gastrectomy on this service since 1922 for recurrent symptoms in whom the primary operation had been performed in other hospitals. They are not included in this review.

It seems to me that this investigation brings out two important points:

(1) A closure of an acute perforation of a gastro-duodenal ulcer (with or without gastro-enterostomy) failed to cure the patient in 39 per cent. of the cases.

(2) In those cases which were subjected to a subsequent partial or subtotal gastrectomy we found another ulcer on the posterior wall of the duodenum which I believe had been overlooked at the time of the primary operation. We have learned from our experiences in resection of the stomach that multiple ulcers occur in about 50 per cent. of the cases. It is therefore apparent that a simple suture of an acute perforation at the pylorus or in the duodenum may fail to relieve the patients of their symptoms in a large number of cases.

The conclusion must necessarily be drawn from this report, that our conservative measures heretofore applied in acute perforations of gastro-duodenal ulcers fail to effect a permanent cure in a very large percentage of the cases. In view of these results the question arises whether it might not be advisable to treat these acute perforations by a more radical procedure. For many years we used conservative methods for chronic gastro-duodenal ulcers and adopted the more radical procedure of subtotal gastrectomy only after we had been convinced that gastro-enterostomy with or without excision of the ulcer

left many patients in a much worse condition than they had been before the operation.

Kreuter, Schwarz, Bruett, Paul and others have published fairly large series of cases showing that, even in the presence of a peritonitis, subtotal gastrectomy does not increase the mortality, if the patient is operated not later than 6 to 12 hours after the perforation has occurred.

We are not prepared at present to adopt this procedure in the presence of an extensive infection of the peritoneal cavity. After all, partial or subtotal gastrectomy is an operation of considerable magnitude. In spite of favorable statistics advocating this procedure we feel at the present time that the mortality might be increased materially, if partial or subtotal gastrectomy would be adopted as a routine procedure in acute perforations of gastro-duodenal ulcers.

However, it may be advisable to attempt a radical cure in cases which come to operation very early and where the infection has not spread beyond the immediate neighborhood of the ulcer.

Furthermore every patient who has been subjected to the conservative procedure should be watched carefully in the follow-up clinic. If the patient still has marked symptoms six to eight months following the operation he should be subjected to a partial or subtotal gastrectomy.

Thanks are due Doctors Beer, Berg, Elsberg, Lilienthal and Moschcowitz for their kind permission to study patients operated by members of the surgical staff on their services.

BIBLIOGRAPHY

[1] Bruett: Das perforierte Magen-u. Duodenalgeschwuer. Ergebn. d. Chir. u. Orthopæd., 1923, vol. xvi, p. 516.

[2] Eliot: The Clinical Features and Treatment of Acute Perforating Gastric and Duodenal Ulcer. ANNALS OF SURGERY, 1912, vol. lv, p. 546.

[3] Kreuter: Ueber die Zulæssigkeit der primæren Magenresektion bei frei perforierten Magen-Duodenalgeschwueren. Klin. Wochschr., 1927, vol. vi, p. 742.

[4] Lewisohn: Frequency of Gastro-jejunal Ulcers. Surg., Gyn. and Obstetr., 1925, vol. xl, p. 70.

[5] Paul: Zur Frage nach dem Operationsverfahren beim perforierten Magen-Duodenalgeschwuer. Dtsch. Zschr. f. Chir., 1922, vol. clxxii, p. 94.

[6] Schwarz: Ueber die Berechtigung der Magenresektion beim frei perforierten Magen-Duodenalgeschwuer. Dtsch. Zschr. f. Chir., 1926, vol. cxcvii, p. 156.

[7] Stenbuck: Causes of Death Following Operations for Perforated Gastric and Duodenal Ulcers. ANNALS OF SURGERY, 1927, vol. lxxxv, p. 713.

ASEPTIC END-TO-END INTESTINAL ANASTOMOSIS

By James K. Shen, M.D.

of Peking, China

from the department of surgery, peking union medical college

There are many reasons why an end-to-end anastomosis of intestine is more desirable than a lateral anastomosis: (1) It reëstablishes the intestinal continuity in its normal anatomical relationship, (2) it does not in the least interfere with the physiology of the restored parts, and (3) the operation is less time-consuming than the lateral anastomosis and can be carried out in a well-nigh aseptic manner.

That there are yet no standard procedures for this very important operation is shown by the large number of proposed methods which have been published in recent surgical literature. A casual review of the subject covering the past five years reveals no less than thirty different methods. Some are obviously too complicated for common practice, while others, calling for manipulations which invite contamination, cannot be considered as "aseptic anastomosis." There are, however, a group of proposed methods which are worthy of comment because of their ingenious nature; these have in common the four following basic principles:

1. The aseptic excision of a segment of intestine.
2. The temporary closure of the ends of the intestine.
3. The abutting and anastomosis of the two stumps by appropriate sutures.
4. The opening of the double diaphragm.

The excision of a segment of intestine is generally done by cautery; where cautery is not available, it is done by knife, followed by the application of pure carbolic acid and alcohol. That the abutting blind ends are best sewed together by properly placed silk mattress sutures is the consensus of opinion of the various authors. The second and fourth principles are intimately related to each other, and it is in their execution that surgeons vary their technic.

Halsted, one of the pioneers in the development of intestinal anastomosis, early practiced the tying-off of the ends of intestine with catgut which was relied upon to melt away promptly. Later, limiting his experiments to the colon, he had the double diaphragm cut by means of a knife inserted by rectum.

Highsmith looped the ends of intestine by linen or catgut ligatures which were held taut by means of specially devised loop-clamps. After completion of the anastomosis, the loops were cut in one leg and then pulled out. Burns employed a ball over which the blind ends of the intestine were sutured. The removal of the ball through the intestinal lumen necessarily broke the purse-string sutures which tightened the ends. Collins suggested placing clamps over retention loop ligatures which had first been held taut. After completion of the anastomosis, the clamps were removed and the loop ligatures pulled out.

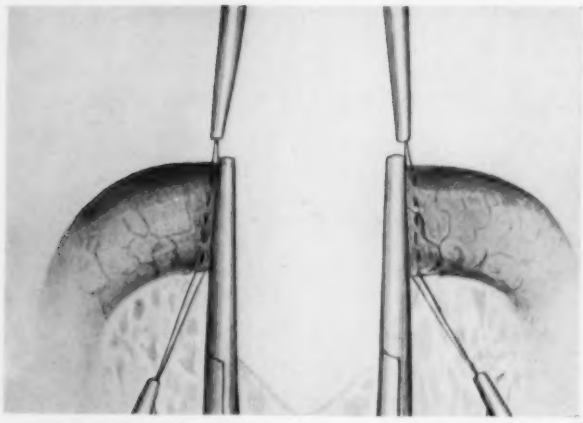


FIG. 1.—A segment of intestine has been excised. The purse-string sutures have been placed. The two Halsted clamps at the antimesenteric borders are holding together the cut ends of the purse-strings.

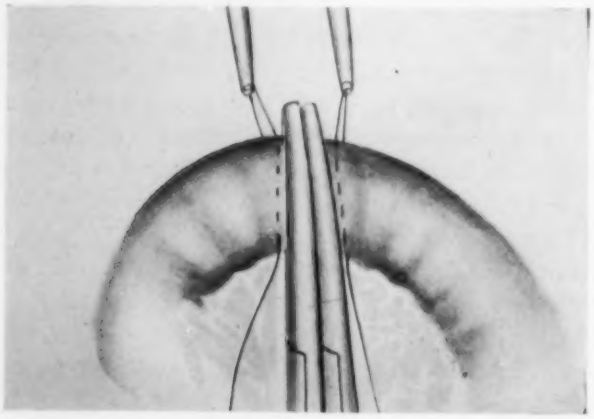


FIG. 2.—Same as Fig. 1 with ends of intestine abutting.

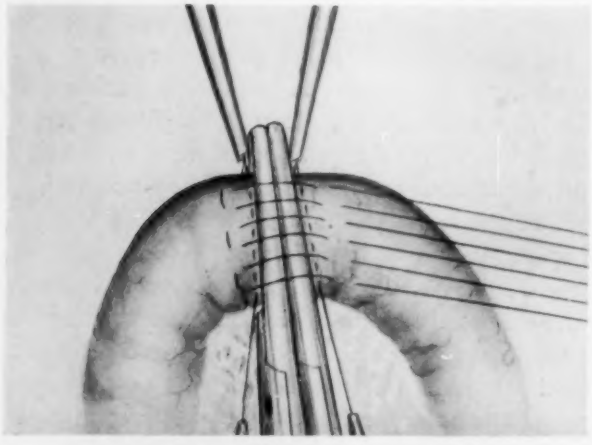


FIG. 3.—Placing of mattress sutures on one side.

Horine had a very ingenious method of loosening a purse-string suture. He placed a free hand string of linen thread between the gut and the first knot of the purse-string suture, and another between the first and second knots. With the pulling of the two releasing strings, the knots of the purse-string sutures were loosened.

Fraser and Dott of Edinburgh and Webster of Peking then developed independently what the writer thinks is the best and safest method for aseptic end-to-end anastomosis of intestine. Both methods called for a specially devised knife which was threaded onto the purse-string ligature, and which cut it upon completion of the anastomosis. The specially devised knife, Fraser and Dott named "the ligature guillotine" and Webster "the revolving tubular knife."

The latest significant contribution to the methods of aseptic end-to-end anastomosis was made by Scarff of Baltimore. He closed the ends of intestine by crushing them, no purse-string being employed. Scarff's method is undoubtedly the easiest and simplest,

if the ends can be relied upon not to open at a critical moment.

The writer does not claim entire originality for his technic, which is largely based upon the methods of Webster, Fraser and Dott. It differs from theirs, however, in the important respect that no specially devised instruments are needed. Thus the operation can be carried out in any small hospital and at any time when there is need of such a drastic measure as partial excision of the intestine.

Technic.—The abdomen is opened in the usual manner and the desired segment of intestine delivered outside the wound. The two points of section having been decided upon, the mesentery is detached to about one centimetre beyond these points. Hæmostasis is carefully secured by ties or transfixing sutures. Two straight Kocher clamps are then applied to each end of the segment to be excised. Excision is done by electric cautery between clamps. The clamps holding the ends of intestine are further "cooked" by the cautery for 15–20 seconds. Beginning from the mesenteric border of one

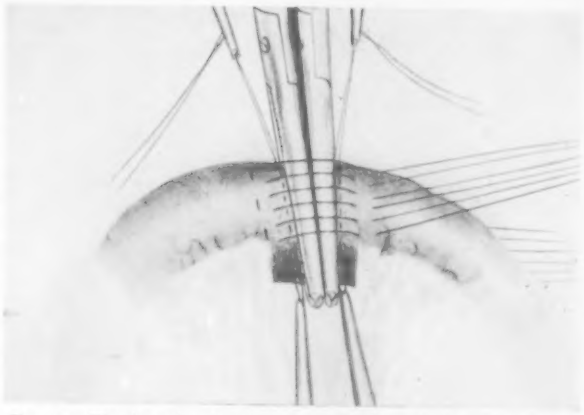


FIG. 4.—The intestine has been turned. The placing of mattress sutures on the opposite side has been completed.

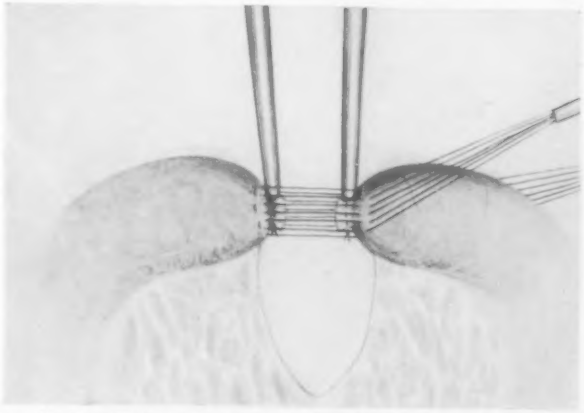


FIG. 5.—Kocher clamps removed and the purse-string sutures tightened. The ends are now blind. The Halsted clamps are still holding the cut ends of the purse-strings.

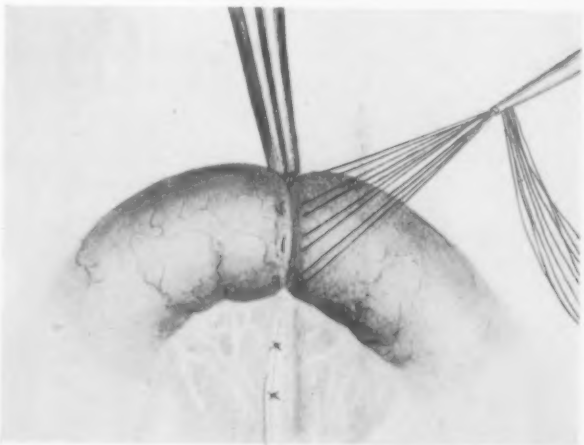


FIG. 6.—All mattress sutures tied. The blind ends are in perfect apposition except at the point where the two Halsted clamps protrude.

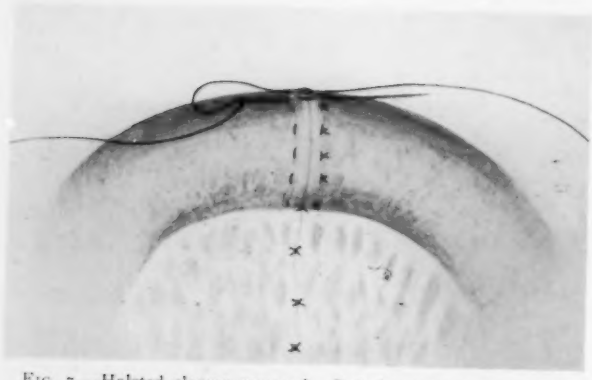


FIG. 7.—Halsted clamps removed. Reënforcement sutures are being applied.

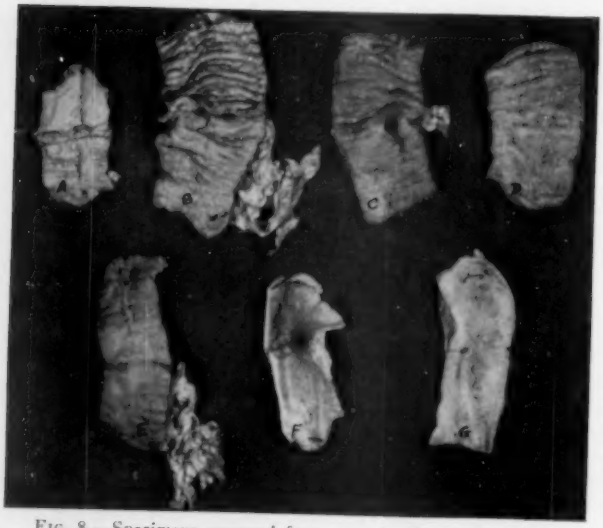


FIG. 8.—Specimens removed from experimental dogs. A, Right after completion of anastomosis. B, One week after operation. C, Two weeks. D, Three weeks. E, Four weeks. F, Five weeks. G, Six weeks.

FIG. 9.—Microphotograph of section 1–10 showing the diaphragm. Immediately after anastomosis.

end, a median silk purse-string suture is placed close to the clamp. The suture is intended to catch the submucous layer, but without entering the lumen of the intestine. The segment of the purse-string suture as it appears at the anti-mesenteric border is then picked up and cut. The cut ends are immediately clamped together by an ordinary straight hæmostat (Halsted) so that the purse-string effect is still intact. The other end is similarly treated. (See Fig. 1.)

The two Kocher clamps are then brought into apposition (Fig. 2) and a row of medium silk mattress sutures are properly placed, first on one side and then on the other. These sutures are also placed through the submucous layers, and as close to the purse-string as possible. Care must be exercised not to catch the purse-string in the placing of mattress sutures. (Figs. 3 and 4.) As the assistant releases the Kocher clamp, the operator steadily tightens the purse-string and ties a double knot at the mesenteric border. The knots are cut short. (Fig. 5.)

The two blind stumps are then held in intimate

apposition by the hands of an assistant; while those of another assistant steady the Halsted hæmostats which are holding together the tight purse-strings. The mattress sutures are then held taut and tied one by one. The segments are now in perfect apposition except at the antimesenteric border where the two Halsted clamps protrude. (Fig. 6.)

These clamps are subsequently removed and a mattress suture immediately placed to cover over the defect. The removal of the two Halsted clamps releases the two purse-string sutures of the blind ends. Gentle pulling of the traction sutures in a four-quadrant direction, but mainly in the two lateral directions, will readily bring open the intestinal lumen at the junction of anastomosis. A few reenforcement sutures in the form of Lembert or mattress sutures here and there now complete the anastomosis. The divided mesentery is sutured by interrupted fine silk sutures. (Fig. 7.)

Aseptic end-to-end anastomosis according to the writer's technic has been done a dozen times on dogs. None of the dogs died as result of the oper-

FIG. 10.—Progress of healing. One week after anastomosis.

FIG. 11.—Microphotograph of section 1–10 showing the beginning of healing of mucosa. Two weeks after anastomosis.

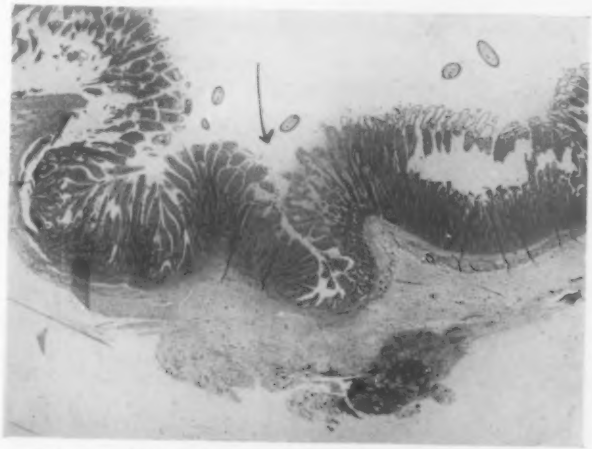


FIG. 12.—Microphotograph of section 1–10 showing more advanced stage of healing of mucosa. Three weeks after anastomosis.

ation, and all had uneventful post-operative histories. At periods varying from a week to six weeks, these dogs were again operated for excision of the specimens. No peritonitis was found and only slight adhesion in two dogs at the site of anastomosis. All specimens are patent. The double diaphragms had sloughed off, even in the one-week specimen. The silk purse-string sutures are seen in the one-week and two-week specimens working their way out into the intestinal lumen. After three weeks, the healing process was found to be complete. (Fig. 8.)

Summary.—A simple method of aseptic end-to-end anastomosis of intestine requiring no special instruments is presented.

This method has been used successfully twelve times on dogs.

N.B. My thanks are due to Mrs. E. B. A. Macmillan who gave valuable suggestions in the preparation of this paper and to Mr. Pu Chang-hai who made the drawings.

BIBLIOGRAPHY

[1] Halsted, W. S.: Blind-end Circular Suture of the Intestine, Closed Ends Abutted and the Double Diaphragm Punctured with a Knife Introduced Per Rectum. ANNALS OF SURGERY, 1922, vol. lxxv, pp. 356–364.

[2] Duboucher, H.: Technic de l'anastomose termino-terminale de l'intestin grele avec les gras intestine. Presse medicale, 1921, vol. xxix, p. 895.

[3] Foley, F. E. B.: An Instrument and Method for Aseptic Anastomosis of the Intestine. Surgery, Gynecology and Obstetrics, 1923, vol. xxxvi, pp. 836–839.

[4] Highsmith, E. J.: Aseptic Resection of Intestines. Surgery, Gynecology and Obstetrics, 1923, vol. xxxvi, pp. 271–273.

[5] Burket, W. C., and McClure, W. B.: An Aseptic Method of Intestinal Anastomosis, An Experimental Study. Surgery, Gynecology and Obstetrics, 1922, vol. xxxvi, pp. 816–819.

[6] Horine, C. F.: Aseptic Technic for the Resection of Intestine, Blind End-to-end Anastomosis with the Release of Purse-string Suture After Anastomosis Is Complete. ANNALS OF SURGERY, 1922, vol. lxxvi, pp. 745–754.

[7] Collins, F. K.: Aseptic Resection of Intestine, End-to-end Anastomosis and Immediate Restoration of Lumen by Use of Special Application of Removable Looped Ligature. ANNALS OF SURGERY, 1922, vol. lxxvi, pp. 739–744.

[8] Fraser, J., and Dott, N. M.: Aseptic Intestinal Anastomosis. British Journal of Surgery, 1923–1924, vol. xi, pp. 439–454.

[9] Carossini, G.: Sur une nouvelle methode d'entero-anastomose termino-terminale au moyen d'une loline demontable. Presse medicale, 1925, vol. xxxiii, pp. 541–543.

[10] Webster, J. P.: Aseptic End-to-end Intestinal Anastomosis. ANNALS OF SURGERY, 1925, vol. lxxxi, pp. 646–669.

[11] Burns, J. G.: Aseptic End-to-end Anastomosis of the Intestine. ANNALS OF SURGERY, 1925, vol. lxxxi, pp. 670–673.

[12] Roeder, C. A.: Improved Technic for Intestinal Anastomosis. ANNALS OF SURGERY, 1924, vol. lxxx, pp. 939–943.

[13] Burket, W. C.: Studies in Aseptic Intestinal Anastomosis. Archives of Surgery, 1924, vol. ix, pp. 530–536.

[14] Rosahoff, W. F.: Blind-end Circular Suture of the Intestine (Halsted Method). Johns Hopkins Hospital Bulletin, 1925, vol. lxxxvi, pp. 81–93.

[15] Scarff, John F.: Aseptic End-to-end Suture of Intestine. ANNALS OF SURGERY, 1926, vol. lxxxii, pp. 490–495.

[16] Perret, C. A.: Technic of New and Strictly Aseptic Method of Enteroanastomosis. Surgery, Gynecology and Obstetrics, 1927, vol. xliv, pp. 378–389.

ACUTE INFLAMMATORY OBSTRUCTION OF THE TERMINAL ILEUM

By Clyde A. Roeder, M.D.
of Omaha, Neb.

In the *Boston Medical and Surgical Journal* for May, 1913, David Cheever described the acute inflammatory obstructions of the terminal ileum which sometimes follow an operation for acute suppurative appendicitis with rubber or gauze drainage. I think he was the first to describe this condition, since I have been unable to find any other description prior to his publication. Most surprising to me is the almost total lack of recognition of his article and the condition he described. Just previous to his publication, I had experienced several similar examples, but was then unaware of the exact pathological anatomy. Fortunately, however, I had recognized the pathological physiology and reported three cases, with recoveries, in the *Boston Medical and Surgical Journal* for August, 1913. Cheever's method of relief was the performance of an ileostomy through the incision previously made for the acutely inflamed appendix. Owing to uncontrollable circumstances, *i.e.*, poor surgical environment and a more limited experience at that time, I, fortunately, was forced to adopt an easier surgical procedure, *i.e.*, an ileo-sigmoidostomy—which I have followed ever since.

During the past fifteen years there has gradually, but too slowly, crept into the minds of the profession the idea that the ileus due to intraperitoneal infections, is as potent an agent in the production of fatalities as the primary peritoneal sepsis. During this time, intestinal drainage operations, including the popular jejunostomy, have been presented and practiced with no small degree of success. It is well to keep in mind that an inflammatory obstruction of the small intestine, rarely the colon, frequently occurs in the presence of septic intraperitoneal exudates and often more rapidly dominates the clinical picture and more rapidly hastens the mortality. It is also well to keep in mind that distention, particularly in the left lower portion of the abdomen, with vomiting and signs of dehydration, coming on forty-eight to seventy-two hours following an operation for purulent appendicitis with peritonitis indicates, in most instances, an acute obstruction of the small intestine. I feel confident in stating that the failure to recognize this complication and to apply early, proper surgical relief, is one of the main reasons for our high mortality rate following operations for acute suppurative appendicitis complicated with purulent peritonitis.

Since 1910, I have operated upon twenty-one patients with inflammatory obstructions of the terminal ileum due to purulent appendicitis and peritonitis and have had only one mortality. This record prompts me to make this report, owing to the high mortality rate still persisting following opera-

tions for the purulent appendix and the even higher mortality rate following all operations for intestinal obstruction. Twenty out of this series were due to the purulent appendix and one to an infected right Fallopian tube. The one fatality was quite likely due to the fact that I applied surgical relief too late. In this series, the youngest patient was an infant two years old and the oldest, a man in the seventies. The histories in all were, singularly, quite similar, excepting in the one case with the acute salpingitis which had not been operated upon previously to the operation for the relief of the acute obstruction of the small intestine. In the twenty cases of acute post-operative obtruction of the terminal ileum due to appendiceal and peritoneal sepsis, the left side of the abdomen first showed distention, with cramp-like pains, starting forty-eight, seventy-two, and up to 144 hours following the operation. As the distention increased, more frequent vomiting and signs of dehydration followed. The relatively low temperature and the lack of rigidity on the left side all pointed against an advancing peritonitis. The pulse rate increased rapidly, but the already elevated temperature did not corollate and the leucocyte count never proved of any value. In my experience leucocytosis generally accompanies an acute intestinal obstruction in its early stages. It is needless to state that satisfactory evacuation of the bowels per rectum was absent, particularly the failure to pass gas. The clinical picture is generally easily recognized after the first experience. When once recognized, the application of immediate surgical therapy should not be delayed, since the patient has two highly fatal lesions—peritonitis and intestinal obstruction, the latter always fatal if not relieved.

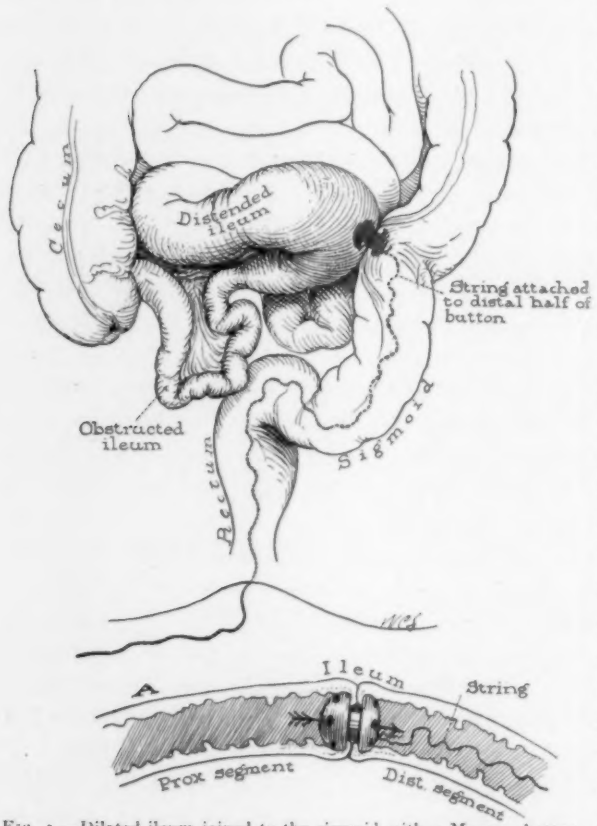


FIG. 1.—Dilated ileum joined to the sigmoid with a Murphy button. Note the string attached to the distal portion of the button.

In the first case of my series of twenty-one, I made an exploratory incision through the left rectus in a field well away from the area of previous operation. In every case following I used the same procedure and was very

much impressed, in most instances, with the almost total absence of purulent fluid in the left side of the abdomen. The terminal ileum was always found greatly dilated down to an area in the right pelvis where a plastic exudate was found sharply kinking and constricting the terminal portion. The most distal portion of the dilated ileum was joined to the sigmoid in each instance and in only two cases, as far as I have been able to determine, did this anastomosis leave the function of the ileo-colonic tract disturbed. In the infant of two years, with acute purulent appendicitis, a troublesome diarrhœa in spells kept up for at least one year after the operation. I was unable to trace this patient after that. In the female with acute salpingitis, an arthritis developed about one year after the operation, which may or may not have been due to ileal absorption of colonic contents.

The main objection to this operation is the possibility of a circling of ileal contents through the colon after the patient has completely recovered; such as occurs sometimes following the ileo-sigmoidostomy of Arbuthnot Lane for colonic stasis. One might well bring up the point that a jejunostomy is more satisfactory, and I have no inclination to dispute the relative merits of this procedure as compared with a direct ileostomy.

In two instances I used a suture technic for the anastomosis, both cases being recognized early and the time element, under local anæsthesia, was not quite so important as in the remaining nineteen cases. In the first three patients operated upon, a Murphy button was used and was passed via the rectum in a few days. In the fourth case the button passed onward into the distal ileum, becoming obstructed at the point where the small intestine was constricted by the inflammatory exudate. It was necessary for me to remove this button two months later through a mid-abdominal incision. In the sixth case the button became lodged in the terminal ileum as in the fourth, but about six weeks later worked its way through the obstructed area in the ileum, finally being passed by rectum. With this experience in the fourth and sixth cases, I decided to attach a heavy silk or linen cord to the distal half of the button in order to obtain, if possible, traction sufficient to compel the movement of the button in the desired direction. Since adopting this method, each button has, in varying lengths of time, passed into the sigmoid. In about half of the instances the cord protruded through the anal canal where additional traction was easily applied. I have used the button with a string attached in an adult following a resection of the small intestine with satisfactory results.

I again wish to state that I feel that suture anastomoses are more desirable than the use of mechanical appliances; but when the patient is desperately ill with an acute obstruction of the small intestine following almost immediately upon an operation for purulent appendicitis and peritonitis, the most rapid method is desirable. With the button one can easily finish this operation in five to ten minutes, and with this modification, its use, with an emergency ileo-sigmoidostomy, leaves very little more to be desired relative to the immediate results.

FIBROMATA OF THE MESENTERY

By William Edgar Darnall, M.D.

of Atlantic City, N. J.

The early history of solid tumors of the mesentery is well recorded by Harris and Herzog,[5] who collected fifty-six cases from the literature up to 1897 and reported one of their own. Of the solid tumors of the mesentery fibromyomata seem to be the most rare.

Greer [4] in 1911 reported a case and reviewed the literature for the last ninety years. He was able to find only thirty-two cases of mesenteric fibroids. His article gives instructive details of them all. His own and the thirty-two others were of a fibromatous nature. Cysts of the mesentery, while not common are about four times more common than solid tumors.

Bevan,[3] 1918, removed a large tumor growing from the mesentery in the cæcal region by clamping and cutting its broad pedicle, only to find, to his dismay, that in cutting away the tumor he had opened the cæcum and ileum, both of which were adherent to its under surface. He was compelled to carry out a resection of the damaged bowel. The tumor on examination proved to be a fibroma. Solomons [8] in the same year recorded a case which he described as fibrosarcoma.

Judd and McVay [9] of the Mayo Clinic, 1920, reported a case of fibroma of the mesentery and brought the literature up to that date. There have been few cases recorded since.

Rawls [10] had a case in which there was a cavity in the tumor mass communicating with the small intestine by a fistula which had ulcerated through from the attached surface. The cavity was filled with foul smelling substance made up of food, blood clots, etc. He discounts the question of its origin being a diverticulitis, since the mass lay entirely within the leaves of the mesentery. The cavity was not mucous lined and the section showed fibroid tissue with few blood-vessels.

In the case of McCauley [11] the tumor and attached bowel was resected. The tumor in gross section resembled a uterine fibroid. It was so intimately related to the intestinal wall that it appeared to have its origin in the bowel musculature. Kyle [12] reported a case which was very vascular and suggested sarcoma but proved to be simple fibroma. DeCourcy [13] reported a case and J. F. Baldwin [14] had a very interesting case in a colored woman, which weighed twenty-five pounds and which he claims to be the largest case of this kind on record.

Always in the rare things in medicine there must be a considerable number of cases which occur but which are never reported and it seems a duty, therefore, when one meets in his experience one of the rare cases to record it. The case submitted herewith brings the reported cases up to 41 in number.

Case Report.—T. D., a young married colored woman of twenty-five years, mulatto in type with a practically negative family history and with no clinical history other than some of the infectious diseases of childhood and a few colds, noticed an enlargement of the abdomen. She has had some pain in the epigastrium, a dragging feeling and attacks of nausea and vomiting and she has lost weight. She has had no children or miscarriages, her menses appeared at the age of fourteen and have been regular, without pain and lasting about seven days. She does not suffer from leucorrhœa. Her pelvic organs are normal. There are no tumors, cysts, disease of the Fallopian tubes or ovaries. Her urine is acid specific gravity 1022 and without casts, but there are numerous pus

cells and some albumen. Her blood shows a negative Wassermann, 4,700,000 reds, 11,800 whites, polymorphonuclears 68 per cent. and hæmoglobin 90 per cent. Her appetite is good, bowels regular and she states that her general health has always been good.

The abdomen was opened in a midline incision. The pelvic organs, stomach and upper abdomen showed nothing abnormal. There was a tumor mass measuring 21 x 18 cm. lying between the leaves of the mesentery, of the ileum about ten cm. from the ileocæcal junction. There were no adhesions, the tumor was movable, but presented on its surface numerous large dark veins. The tumor mass with about thirty inches of ileum was removed. An anastomosis of the ileum was done and the appendix removed. The patient went into shock and died about eight hours after the operation.

Pathological report by Dr. Robert Kilduffe.—Specimen consists of a large mass measuring 21 x 18 cm., of fibrous tough consistency. There is attached to this and hugging it closely, thirty inches of small intestine. On section the mass is dense, firm and has a somewhat fatty appearance. Its shape is roughly globular. It has the general appearance of being encapsulated. A large portion of adherent omentum accompanies the specimen. Microscopically the histology is that of a fibroma in which there are a moderate number of small vessels which are more or less congested. There is no histological evidence of syphilis, tuberculosis, or malignancy.

Quoting from Judd[9]: "The origin of mesenteric tumors offers many interesting hypotheses, but has been very little discussed by any of the writers on the subject. Greer[4] states that fibromas develop in the connective tissue, grow slowly and rarely recur after complete removal. Royster[6] believes that all such tumors are of mesoblastic origin, solid or cystic and that the solid tumors may be either benign or malignant."

The other theory is that these tumors originate in the uterus as fibroids, become migratory in character and attach themselves to the mesentery. Benign migratory tumors rarely affect the essential circulation of the part to which they may become attached. Together with the fact that no other fibroid tumors were found in the uterus and that this tumor was confined between the leaves of the mesentery it is more probable that this tumor grew *in situ*.

The diagnosis of mesenteric tumors is rarely made before the abdomen is opened and is impossible in most cases. The mortality is high. Vance[7] reports a mortality of 46 per cent. where resection of the bowel was done and Begouin's[1] mortality was 52 per cent.

BIBLIOGRAPHY

[1] Begouin quoted by Greer: Brit. Med. Jour., 1911, p. 1085.
[2] Bevan, A. D.: Surg. Clin., Chicago, 1918, vol. ii, p. 479.
[3] Bowers, L. G.: Annals of Surgery, 1906, vol. lxiv, p. 892.
[4] Greer, W. J.: Brit. Med. Jour., 1911, vol. ii, p. 1085.
[5] Harris and Herzog: Annals of Surgery, 1897, vol. xxvi, p. 66.
[6] Royster, H. A.: Jour. Am. Med. Assn., 1911, vol. lvii, p. 531.
[7] Vance, J.: Annals of Surgery, 1906, vol. lxiii, p. 366.
[8] Solomons: Medical Press, Oct. 2, 1918.
[9] Judd and McVay: Surg., Gynec. and Obstet., 1920, vol. xxxi, p. 372.
[10] Rawls: Va. Med. Monthly, 50764, Feb. 1923.
[11] McCauley, Chas.: Medical Press, London, pp. 119, 299, April 15, 1925.
[12] Kyle, H. G.: Brit. Jour. Surg., 1921, vol. xiv, p. 295.
[13] DeCourcy, Jos. L.: Surg., Gynec. and Obstet., vol. iii, No. 40. March, 1925.
[14] Baldwin, J. F.: Am. Jour. Surg., vol. ii, p. 160, Feb., 1927.

LYMPHANGIOMA OF OMENTUM

By William H. Fisher, M.D.

of Toledo, Ohio

surgeon to st. vincent's hospital

Lymphangioma is a tumor composed of lymph-vessels. It is therefore an organoid structure consisting of endothelial cells and supporting connective tissue, both of which are involved in the neoplastic process. Lymph-nodules or foci of round cells are often present to complete the parallel with a lymphatic structure.

The very early stages and exact origin of these tumors have not been demonstrated, and as Hedinger points out, they require differential diagnosis from a variety of other abdominal cysts. Their neoplastic nature seems assured since the walls contain cellular connective tissue, often much smooth muscle tissue, and lymph-follicles. After partial removal the remaining portion may rapidly increase in bulk. Along the edges of the growths, Sick found proliferating areas of cavernous lymphangioma. He assumes that they arise from misplaced and embryonal islands of connective tissue and lymph-vessels. In the omentum and mesentery of newborn cats and pigs, Ranvier has demonstrated such misplaced islands of tissue.

In the origin of lymphangioma it must be assumed that there exists a local predisposition resulting from an embryogenic disturbance similar to that assumed for hæmangioma. Of the nature of this disturbance nothing is definitely known, but the congenital origin of most lymphangiomas is a striking feature in their etiology. A partial isolation of a segment of lymph-vessels with imperfect development and retention of abnormal powers of growth may be supposed to exist. New formed lymph-vessels are also present in many benign and malignant tumors, especially with endothelioma and sarcoma.[1]

Adami in considering cavernous lymphangioma states that he finds extensive overgrowth, with fibrosis of the parts between the dilated lymph spaces. The conditions are all congenital, and we must conclude that there is obstruction to onward flow of the contained fluid, due to some abnormal relationship of the different vessels.

These cysts are lined with endothelium, containing clear lymph, and having fibroid walls. Many of the large cysts appear to be absolutely closed off, not communicating with their neighbors. We must suppose that, with increasing distention, there has been a valve-like closing of the channel of which they are a dilatation, that the endothelium has grown *pari passu* with the dilatation, and that this endothelium has secretory powers. The mere force of the lymph flow cannot explain such extreme development; we have to assume active excretion, which, indeed, is indicated by many other considerations

and actual experiments of Heidenhain.[2] He further differentiates telangiectatic cases, such as cervical hydrocele, sacral hygroma, growth of lymph channels *pari passu* with other changes in tumors or malignancy, from true lymphangiomas.

Some fifty cases of omental cysts are reported in the surgical literature. About 50 per cent. are in adult life. Of these, the majority are in the female; many of these are of the secondary variety, subordinate to tumor formation and cannot be considered as true primary lymphangiomas. The other 50 per cent. of reported cases are under ten years of age and are considered embryonic.

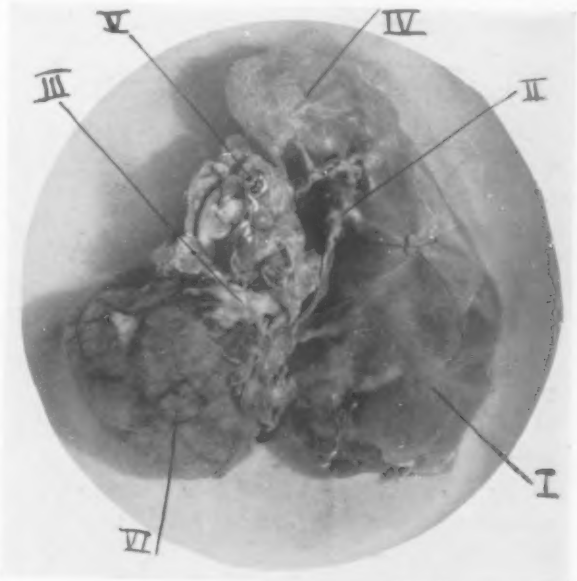


FIG. 1.—1. Large thin walled cyst—multilocular. 2, 3. Hyperplastic lymphoid tissue. 4. Thickened portion of cystic wall. 5. Inflamed and contracted omental remnant. 6. Multilocular cyst with firm fibrous wall.

But few, if any, subjective symptoms are present, except those due to inflammation or torsion of cysts with resultant hemorrhage and inflammation. Pean describes three points in diagnosis: superficial location, abdominal passive mobility with downward movement limited, and absence of functional disturbance.[3] Hearn and Arzela diagnosed omental cyst; Brandt a large omental cyst by exclusion; Stillman an omental cyst or ovarian cyst with long pedicle.[4, 5, 6, 7] In differential diagnosis, tuberculous peritonitis, ascites, and ovarian cysts must be considered.

Hasbrouck claims a 6 per cent. mortality.[8] Extirpation of primary lymphangiomas is the only method of cure. Tapping of cysts, secondary to tumor formation, has been reported successful after excision of tumors. Removal of malignant growths with complete omenectomy for transplantation omental cysts, and followed by intensive X-ray, has not prevented recurrence and ultimate death of three cases in the writer's experience.

CASE REPORT.—Edwin D., age four, was admitted to St. Vincent's Hospital suffering from acute abdominal pain. Family history is negative. Personal history is negative, except infected tonsils. Pale, well nourished, a pendulous abdomen had been noted lately and ascribed to an appetite above the normal.

Three days before admission he complained of abdominal pain and distress, increasing in severity and paroxysmal in type. Examination revealed a large abdomen with numerous dilated veins, a hard mass in the upper left quadrant, pain felt on pressure and reflected to left inguinal region and causing marked flexure of both thighs. The

rest of abdominal cavity filled with a large, flaccid, fluctuating cyst. Pulse and temperature ranged around 100. Examination of blood shows Kahn and Wassermann and repeated tests for filiaria Bancrofti were negative. Red 4,296,000; white 13,050; polymorphonuclears 75 per cent.; lymphocytes 21 per cent.; mononuclears 2 per cent.; transitional 1 per cent.; eosinophils 2 per cent.; slight microcytosis noted. Examination urine practically normal, except 2 + acetone. White blood-cells and mucus. Chyluria as well as chylorrhœa negative.

FIG. 2.—Wall of large lymphatic cyst of omentum, lined by flattened endothelium, beneath which there is a zone of inflammatory fibroblastic tissue. Beneath this, in the omentum, are dilated lymph vessels. (Cystic lymphangioma.)

Operation.—Under ether anæsthesia February 17, 1927, upper right rectus incision disclosed a large multilocular cyst, filling the abdominal cavity and attached to the hilus of the spleen, one-half of gastro-colic omentum and splenic flexure of colon. At this point rotation of one large and several smaller cysts had produced circulatory obstruction with resultant inflammation and hemorrhage in cyst cavities. The normal gastro-splenic omentum and one-half of the gastro-colic were replaced by a thickened dark, grayish, fibrous tissue. This tissue extended from over the right of the median line to the left in its entirety. One large cyst filled the entire upper and lower right quadrant, the second largest the lower left, the third cyst with numerous smaller ones, radiating along the course of the blood-vessels had rotated at the splenic flexure of the colon and filled the upper left quadrant. This entire mass with its fluid contents weighed twelve pounds.

The operation consisted in the complete extirpation of the cyst bearing omentum, dividing it longitudinally beyond the line of fibrosis of gastro-colic omentum, and dissecting closely from the under surface of colon in its entirety to the left. (Fig. 1.) The incision was closed without drainage. Convalescence was uneventful and patient discharged March 9, 1927. At present writing no evidence of recurrence and patient in perfect health.

Pathological Report by Dr. Thomas L. Ramsey. Tissue taken from the cyst wall shows a cyst lined with flattened endothelium. Beneath the endothelium there is a narrow zone of inflammatory fibroblastic tissue continuous with the tissue of the omentum. Throughout the adipose tissue of the latter are numerous irregular sinusoidal or cavernous dilated lymphatic vessels, lined with flattened endothelium and containing lymph. Throughout the adipose tissue, and especially around the cystic lymphatics there is a diffuse inflammatory infiltration and fibroblastic proliferation of slight degree. In the wall of

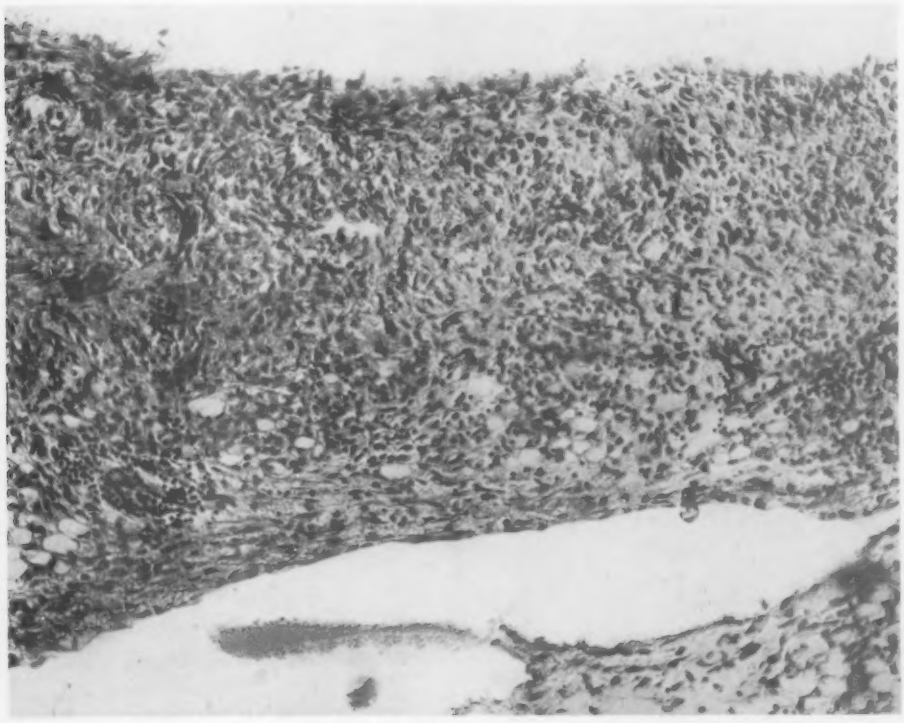


FIG. 3.—Higher power photomicrograph of portion of wall of lymphatic cyst of omentum, showing flattened endothelium lining wall of large cyst, fibroblastic zone beneath this, and portion of smaller lymphatic cyst below. Unstriped muscle cells in cyst wall; in some places these appear in definite bundles.

the larger dilated lymph-vessels small bundles of unstriped muscle are present; and occasionally a small group of lymphoid cells. (Figs. 2, 3, 4, 5.) Pathological Diagnosis.—Cystic lymphangioma (chylous cyst); congenital, lymph stasis. Not a true neoplasm, but a congenital disturbance of development. Inflammatory changes secondary due to pressure. No malignancy.

Dowd analyzed thirty-seven collected cases in 1917. Of these five, and possibly another, microscopic findings show endothelium of cyst wall; two had epithelium; fifteen had fibrous tissue; eighteen dark-colored fluid.[9]

Rodman reported cyst of sixty pounds; Ormsby one of seventy-five pounds filled with fluid too thick to flow; Karas one associated with pyæmia with ciliated cells lining cyst wall.[10, 11, 12]

Omental cysts associated with uterine myomæ reported by Outerbridge and Girvin. Both characterized by fibrous wall and endothelium. In both inflammation and adhesions produced lymph stasis.[13, 14]

Funk reports cyst containing three gallons of fluid, fibrous tissue and endothelium.[15]

Pybus, case age four, football in size; no epithelial layer, and peeled from omentum.[16]

Arzela, case three years old, cyst contained one and one-half litres of transparent fluid, and believes the cause is an anomaly of development of a

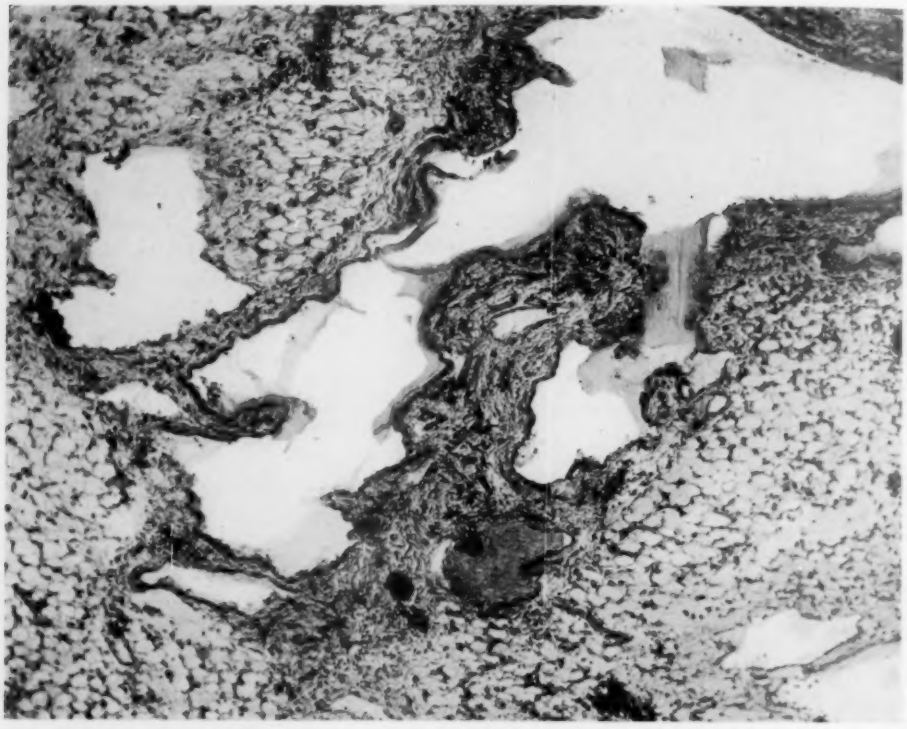


FIG. 4.—Cavernous lymphatic vessels in omentum. "Cystic lymphangioma." Lymph stasis. Slight inflammatory infiltration of omental adipose tissue.

lymph gland in the sense of deficient proliferation of the mesenchyma which constitutes the septa, this deficiency substituting a cystic cavity with lymph contents for the lymph gland.

Such a mechanism of development would explain the formation of cysts in all regions rich in lymphatics, viz.: congenital lymph gland cysts originating from lymph glands in their first period of development.[17]

Grausman and Jaffe "28" report, male forty-four, abdominal mass with pain. Many omental cysts lined with endothelium and supported by connective tissue layer.[18]

They consider their case as a "true blastoma from undifferentiated mesenchyma, which is capable of producing lymphatic vessels by proliferation of lymphangio-blasts.

"Many of these newly formed lymphatic vessels become enlarged and cystic, due to the blocking of the outlets and possibly because they are blind

secondary changes such as dilatation and proliferation of the endothelium. They believe that the preformed lymphatics are not involved in the tumor growth."

Comments.—The findings in this case show a primary true lymphangioma of the omentum. The direct cause of cystic development, a fibrosis of the gastro-colic and gastro-splenic omentum. What caused this fibrosis is indeterminate.

FIG. 5.—Higher power photomicrograph of portion of wall of cystic lymph vessel in omentum, showing lymph content about the wall and endothelial lining.

The history of case is negative except chronic infective tonsillitis. No abnormalities existed in the thorax that would have a bearing on this cystic development of omentum, such as tumors in the mediastinum, or obstruction of thoracic duct due to inflammations, thrombosis of the left brachiocephalic vein, or of tricuspid insufficiency, which would produce backward pressure of blood in the subclavian.

While in doubt as to etiology, should this case be dismissed as one of congenital origin, embryonic rests, malformations, or defects?

Various structures of the body are affected by microörganisms of hæmatogenous origin from different foci of infection.

The effects produced are dependent upon the varying types of organisms of their virulency, of structures involved, and of the resistance of person.

Connective tissue change is concomitant of infection. Chronic infective tonsillitis may involve any organ or structure in the human body.

The supposition arises in this case of the hæmatogenous origin from this foci of infection, the organism having a selective action upon the gastro-colic and gastro-splenic omentum; at first inflammatory in character, the end result of which, the formation of dense fibrous tissue. This fibrosis then producing a stasis and obstruction of the lymph circulation in the omentum, and the consequent formation of three large, and many small, omental cysts. *Pari passu* with the increase of size and weight of the cysts, the pull and traction upon the inflamed gastro-colic and gastro-splenic omentum, tend to increase the development of fibrous tissue.

BIBLIOGRAPHY

[1] Ewing: Neop. Dis., p. 228.
[2] Adami: Prin. Path., p. 821.
[3] Pean: Quoted by Van Hook and Kanaval in Keen's Surgery.
[4] Hearn: ANNALS OF SURGERY, 1897, vol. xxv, No. 1, p. 703.
[5] Arzela: Policlinic, Rome.
[6] Brandt: Quoted by Outerbridge. ANNALS OF SURGERY, vol. lx, p. 680.
[7] Stillman: Quoted by Outerbridge. ANNALS OF SURGERY, vol. lx, p. 680.
[8] Hasbrouck: ANNALS OF SURGERY, 1908, vol. xlviii, p. 206.
[9] Dowd: ANNALS OF SURGERY, November, 1911, vol. liv, p. 617.
[10] Rodman: ANNALS OF SURGERY, 1909, vol. xlix, p. 427.
[11] Ormsby: Med. Press. and Circ., London, 1883, vol. xxv, p. 258.
[12] Karas: Virchowo Archives, Bd. clxxxviii, p. 138.
[13] Outerbridge: ANNALS OF SURGERY, December, 1914, vol. lx, p. 680.
[14] Girvin: ANNALS OF SURGERY, 1921, vol. lxxiv, p. 792.
[15] Funk: Surg., Gyn. and Obs., January, 1914, vol. xviii, p. 70.
[16] Pybus: Lancet, London, 1917, vol. cxcii, p. 63.
[17] Arzela: Policlin., Rome, 1922, vol. xxix, Sez. chir., p. 417.
[18] Grausman: ANNALS OF SURGERY, 1928, vol. lxxxvii, p. 66.

LYMPHOSARCOMA OF THE MESENTERY

By Leslie L. Bigelow, M.D., Ernest Scott, M.D.
and
Sterling W. Obenour
of Columbus, Ohio
From the Department of Surgery and Pathology, College of Medicine, Ohio State University

Tumors of the mesentery are rarely encountered. Julian L. Rawls,[1] in a search through medical reports, has found about two hundred cases of cystic tumors of the mesentery and one hundred cases of solid tumors, to which he adds two cases of his own, one being a tuberculosis of the cæcum, and the other, a fibroma. Harris and Herzog[2] collected fifty-six cases of mesenteric tumor in 1897. In 1905, Vance[3] collected twenty-eight cases which appeared in the literature of the preceding five years, H. G. Kyle[4] reported a case of fibroma of the mesentery in 1921 and, in searching the literature for the past ninety years, found report of only thirty-two tumors of like nature. Lipomas of the mesentery, according to B. A. Alton,[5] are the most common solid tumor of the mesentery. In 1918, Bevan[6] of Chicago reported a huge fibroma of the ileocæcal junction.

In a review of the literature since 1920, we have found reports of twenty-five cases of solid tumor of the mesentery. Of these, eight were definitely sarcomatous in nature, eight were lipomas and in nine cases the exact diagnosis was not definitely stated.

Lymphosarcoma is the most common type of malignant tumor occurring in the mesentery. It is a growth arising in the lymphoid tissue; it is somewhat more restricted locally than is Hodgkin's disease or pseudo-leukemia; it possesses greater invasive tendencies than either of these diseases but is without marked malignancy at least as indicated through the blood stream, according to Nelson.[7] Delafield and Prudden[8] state that the tumor consists primarily of lymphoblasts and agree with Ribbert,[9] the name should properly be "lymphoblastoma." These authors state that in the metastasizing lymphoblastoma characteristically follows the lymph channels, at first spreading along them from one lymph-node or follicle to the next, but finally invading the surrounding structures, until all are fused into one firm mass. It progresses from one region to another without ever becoming generalized as do leukemia or pseudo-leukemia. Metastasis by way of the blood stream is rare. In differential diagnosis it is stated that the lymphoblastoma may be distinguished from the large round-celled sarcoma by the lack of a proper stroma, its early limitations to the lymphatic system, the capillary size of its blood-vessels, the absence of hemorrhage, and its tendency to metastasize in organs ordinarily spared by malignant growths.

Nelson[7] states that lymphosarcoma occurs in males twice as often as in

females; that it may occur from infancy to old age; that the differential diagnosis is extremely difficult and is rarely made, and that the prognosis is uniformly fatal.

CASE REPORT.—T. E. G., male, white, aged eight, admitted to the Children's Hospital on March 5, 1927, showing a marked distention of the abdomen. The family and social history were negative. Three weeks previously the child, immediately after breakfast, was seized with an attack of vomiting, accompanied by abdominal cramps. Nothing could be ascertained upon physical examination, the vomiting being the only indication of abnormality. Six days after the attack the mother noticed some distention of the abdomen which increased daily. The bowels moved with laxatives.

Physical Examination.—Showed an abdomen markedly distended, skin tense, and a few prominent veins over the upper part. Percussion revealed an area of tympany over the epigastrium, the remainder of the abdomen being dull. Dulness shifts slightly with change of position. No succussion sounds are heard and no fluid wave is noted. There was no rigidity. There was a small area of tenderness in the upper left quadrant. No masses were palpable or visible. On rectal examination there was the same feeling of tension that was noted over the abdomen. No tenderness on rectal examination, but a mass was felt high in the left side. X-ray of the chest showed a slight peribronchial infiltration. X-ray of the abdomen showed a possible mass, the size of an orange, to the left of the midline. The urine was negative. The blood picture showed: Red blood-cells, 3,000,000; hæmoglobin, 70 per cent.; white blood-cells, 10,100.

It was thought that the condition might be due to tuberculous peritonitis, but no pre-operative diagnosis was made, and the operation advised was frankly exploratory.

A four-inch median vertical incision was made below the umbilicus. The peritoneum at the upper angle of the incision was thin, but it was quite thick below, suggesting tuberculosis. A small amount of free, odorless, fluid escaped (culture from this showed no growth). An enormous omentum was then delivered. It was thick, had a granular feel and was friable. With the omentum delivered a dumb-bell shaped mass was felt in the upper abdomen which proved to be a tumor of the mesentery, the size of an adult fist. The growth had extended to the wall of the small intestine above. No effort was made to resect, as the entire mesentery of the small intestine was shot full of similar small masses. A small piece of the parietal peritoneum was cut away for examination, much of the omentum was removed and the wound was closed without drainage. The child was put to bed in poor condition, and died within an hour. The operation was classified as "Exploratory laparotomy for sarcoma of the mesentery, small intestine, and omentum." This operative diagnosis was confirmed by the pathological findings which follow:

Autopsy Report No. 3863.—May 7, 1927, surgical service, Dr. Leslie L. Bigelow, pathologist, Dr. Ernest Scott. Upon opening the abdomen the omentum is immediately seen to be greatly increased in amount, and upon closer examination it is seen to consist of its usual thin membrane which is traversed by pale, apparently cellular streaks; these streaks or masses of cells are arranged in a somewhat definite network closely resembling the distribution of the normal fat over the omentum. Upon drawing the omentum forward it is found that not all of the omentum was at first visible but a large quantity was tucked away in the lateral portion of the abdomen and in the pelvis. When the omentum is entirely assembled it forms a large mass 20 x 25 cm. and is unusually friable. Upon examination the intestinal tract is found to be free and of normal appearance with the exception of one area in the jejunum, where there is a distinct nodule measuring 3½ cm. in diameter, involving the larger portion of the intestinal wall; immediately adjacent to this there is a second mass of equal size lying within the mesentery. Upon section the lumen of the intestine is found not to be constricted and it is found that the tumor mass does not involve the mucous surface. The tissues of the tumor are of uniform pinkish color and slightly friable. The mesenteric lymph-nodes are enlarged and

near the base of the mesentery there is a confluent mass of nodes. Upon section these nodes present the same pinkish color and are friable similar to that noted in the tumor attached to the intestines. The liver is of the usual size, but along its inferior margin there are several whitish nodules standing out somewhat conspicuously from the liver substance and measuring 2 x 2½ cm. in diameter. On section through one of these nodules it presents the same appearance and characteristics of the nodules in the mesentery. Upon removal of the liver examination of its surface reveals other and somewhat larger nodules imbedded in its substance, one of these masses of tumor tissue corresponding in size and shape to the gall-bladder, on section presents a yellowish discoloration resembling a bile-stained mucous membrane. The impression is gained that the gall-bladder has been so infiltrated by the tumor mass that its lumen has been almost completely obliterated and only a small portion of the bile-stained mucosa remains. Examination of the kidneys, adrenal glands, ureters and bladder fails to reveal any evidence of pathologic change, or of metastatic nodules. Examination of the spleen and pancreas also give completely negative results. Examination of the thorax is entirely negative. The anatomical diagnosis is sarcoma of the mesentery with metastasis in the omentum, the wall of the jejunum, the liver and the gall-bladder.

Microscopic Examination.—The cell is typical of the lymphoblastic type. Many mitotic figures are present. There is a marked metastasis into the liver. The omentum is thoroughly infiltrated and the intestinal wall is involved in one area, the cells having infiltrated to the submucous layer. There is a retroperitoneal infiltration adjacent to the attachment of the mesentery.

DISCUSSION

The mortality following operations for solid tumors of the mesentery, with or without resection of the intestine, is high. The prognosis of lymphosarcoma is, according to Nelson [7] universally fatal. It is his belief that any surgical treatment is merely palliative. However, according to Bigelow and Forman,[10] early surgical removal of the involved glands may be followed by recovery. It is their belief that when but one leaf of the mesentery is bulged and the intestinal wall is not involved, the tumor mass may be enucleated without dangerous interference with the blood supply. If the tumor mass is more centrally placed, bulging both leaves of the mesentery, or if the intestine is adherent to the growth and involved in the process, resection should be done at once. Harris and Herzog,[2] Sawyer, Mathews, and others, report successful removal of mesentery sarcoma combined with enterectomy.

The case here presented is the second encountered by one of us, the first being in July, 1919. This adds another to the short list of mesenteric sarcomata. It is regional in type, and has no history to which can be attached etiological significance. The case conforms to the established picture of regional lymphosarcoma. The size of the mesenteric tumor, as compared with other involved regions, leaves little doubt that the sarcoma arose in the mesentery.

BIBLIOGRAPHY

[1] Mesenteric Tumors: Virginia Med. Monthly, February, 1924 (Rawls).
[2] Harris and Herzog: Solid Mesenteric Tumors. ANNALS OF SURGERY, 1897, vol. xxvi, p. 66.

[3] Vance, J.: Solid Tumors of the Mesentery. ANNALS OF SURGERY, 1906, vol. xliii, p. 366.
[4] Fibroma of Mesentery (H. G. Kyle): Brit. J. Surg., vol. ix, p. 295, October, 1921.
[5] Lipoma of Mesentery (B. H. Alton): Boston M. & S. J., vol. clxxxv, p. 205, August 18, 1921.
[6] Bevan, A. D.: Surg. Clinics, Chicago, vol. ii, p. 479, 1918.
[7] Nelson: vol. iii, pp. 366–367.
[8] Delafield and Prudden: Pp. 430.
[9] Ribbert: Geschulstlehre, 2nd ed., Bonn, 1914, p. 364.
[10] Lymphosarcoma (L. L. Bigelow and J. Forman): ANNALS OF SURGERY, vol. lxxi, p. 11, January, 1920.

BRACHIAL PLEXUS ANÆSTHESIA

ITS INDICATIONS, TECHNIQUE, AND DANGERS

By Prof. D. Kulenkampff

of Zwickau, Germany

In collaboration with

M. A. Persky, M.D.

of Providence, Rhode Island

From the surgical clinic of prof. heinrich braun, at the krankenstift, zwickau, germany

With the rich clinical experience of Prof. Heinrich Braun showing the safety of novocain-adrenalin solution in local anæsthesia, a new impetus was given its use. Anæsthesia of the trigeminus, median and ulnar nerves was an established possibility; the logical question presented itself; why not a complete brachial plexus anæsthesia to open the way for a vast amount of surgery of the upper extremity without the use of a general anæsthetic? Essentials to this accomplishment were fundamentally two: exact knowledge of the location of the nerve trunks which were to be injected, and the particular avoidance of injury to any of the vital structures in its immediate neighborhood. A careful anatomical study of the trunks forming the plexus showed that the best location to reach them with the needle was in the immediate neighborhood of the subclavian artery where it passes over the first rib, the latter serving as an efficient barrier to deeper penetration by the needle. Here, too, as an added advantage for the site, all the branches of the plexus could be anæsthetized through one injection. The proximity of the subclavian artery gave only mild cause for concern, for one of us (D. K.) has proven clinically on numerous occasions, that a thick-walled artery like the subclavian could be punctured with impunity by a thin needle such as is used in this work. The structure which caused more concern was the large subclavian vein, because of the fact that even a few c.c. of a ½ per cent. solution of novocain injected into a vein so close to the head is quickly productive of a serious novocain poisoning. But the position of the vein, lying as it does, medial and in front of the artery from which it is separated by the lower part of the scalenus anticus muscle, affords it a reassuring degree of protection. (Figs. 1 and 2.) The apex of the lung was another structure which gave no small cause for concern; but again the first rib plays an important part in affording protection to it. Needless to say, a clear and definite mental picture of the anatomy of this region is absolutely necessary to the proper accomplishment of the anæsthesia in question. We have then, as limiting the field in which to work, these structures: the clavicle marking the lower limit, the first rib, the upper, and the subclavian artery (determined by its pulsations) the outer limit of safety. The first attempt at plexus anæsthesia by the route to be described was made by Professor

Kulenkampff on himself. (Fig. 3.) It was by this means proven that the route was a safe one of approach to obtain an anæsthesia for the hand, forearm and arm. Previous attempts at plexus anæsthesia had been made by Hirschel,[3] his route of approach being through the axilla. There, too, the plexus is somewhat superficial and orientation to it gotten by the axillary artery; but the many disadvantages to this approach as well as its failure to anæsthetize many of the branches taking origin above the axilla, failed to secure for it any degree of popularity. Working on this supraclavicular route Perthes[4] attempted to simplify the technic by using a mild electric current to stimulate the plexus when the needle was thought to be in proper position, and thus give proof as to its location. Another modification of plexus anæsthesia came with Mulley's[5] work. He selected a point about three fingers' breadth above the clavicle as the entrance point for the needle. It is made to enter ½ cm. posterior to the external jugular vein and from this point attempts to locate the plexus are made. But in this route the danger of injury to the apex of the lung and to the deeper structures of the neck are very great, as the protecting first rib is not utilized in this method. Hohmeier[6] and Balitzky[7] have devised an infra-clavicular route of approach. In this technique the needle is entered ½ cm. under the midpoint of the clavicle and passes upward and inward; after piercing the pectoralis major the plexus is encountered from 2 to 5 cm. further inward. Still another modification was that of Capelle[8] using the Hirschel technique as a basis. He injects 20 to 30 c.c. of novocain solution in the bicipital sulcus, depositing the solution around the artery. The foregoing is a brief review of the progress made in the work of plexus anæsthesia.

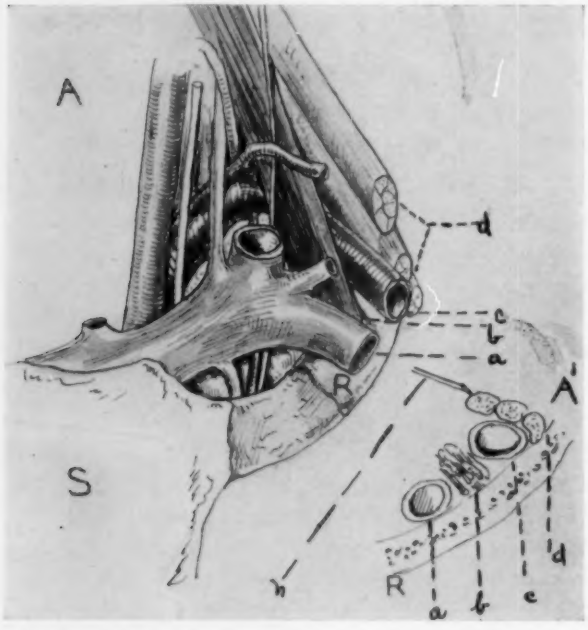


FIG. 1.—A and A': a: subclavian vein; b: scalenus anticus muscle; c: subclavian artery; d: cords of brachial plexus as they cross 1st rib; S: sternum; R: 1st rib; n: needle.

INDICATIONS

Practically all conditions of the upper extremity which necessitate operative procedures, lend themselves to plexus anæsthesia. As the needle in this type of anæsthesia does not enter the infected area, no concern need be felt

for spreading the infection if it is a pus case. In general, one can say that every injury of this region, whether mechanical or bacterial, is suited for plexus anæsthesia. It is especially valuable in the numerous hand and forearm injuries to which the diversified callings of the worker expose him, such as amputations, disarticulations and fractures. Operations on tendons and tendon sheaths offer a major field for the application of this method. In shoulder dislocations it is invaluable. As in local anæsthesia in general, a patient who stamps himself as unfit by his general reaction to the preliminaries attending the actual anæsthesia, is best operated on under a general anæsthetic. In some minor operations of the hand or forearm where the type of injury is slight, it is not worth the time or energy to do a brachial block; such cases are best treated either by a localized injection or under a general anæsthetic.

FIG. 2.—The plexus and related structures (after Corning).

TECHNIQUE

A *sine qua non* to a proper technique is a clear mental picture of the relation of the structures as they lie under the skin, and a keen sense of direction in which the needle is to be pointed; this is such, as if one wished to strike the tip (spinous process) of either the second or third dorsal vertebra. (Fig. 6.) The point of the needle's entrance is over the midpoint of the clavicle, just external to the external jugular vein; the latter can definitely be brought into view by asking the patient to close his lips and puff his cheeks. Finally came for decision the strength of solution to be used, and the possibility of injury to the nerve trunks by the penetrating needle. Both of these considerations could be definitely answered through the accumulated experience which this hospital has afforded. As to solution: a 2 per cent. novocain-adrenalin was found the most effective; and as for nerve trunk injury, at no time have any clinical signs of permanent nerve injury been found as a result of this type of anæsthesia. The kind of needle used has been of great importance. We have used one 35 mm. long and .5 mm. thick; occasionally one 45 mm. long and .6 mm. thickness has been found practical. The injection is best carried out with the patient in the sitting posture, an ordinary chair suffices. The arm on the side on which the plexus is to be anæsthetized lies quietly in the lap with the shoulder relaxed. (Figs. 4 and 5.) This position not only permits of an easy approach to the plexus, but maintains a certain degree of tension

on it, which helps in its location by the entering needle. Should the patient's condition prevent his sitting up, he is allowed to lie in bed with a pillow under his shoulder blades and his head turned to the side opposite to the one on which work is being done. The operator seats himself on a stool adjusted to a height which will make him most comfortable. The comfort of patient and operator is important, as plexus anæsthesia is often time-consuming. Before beginning the actual anæsthesia the patient is told that he must immediately notify the operator when the plexus is touched by the needle. He will recognize this by experiencing the same sensation as when striking his "crazy" bone. This pain can be demonstrated to the patient by pressure on the ulnar nerve in the sulcus between the humerus and ulna; in thin people, the supraclavicular portion of the plexus can be rolled under the finger with its consequent tingling sensation in the fingers. It is for this type of sensation that the patient is cautioned to watch and report immediately by saying "Now." The patient must also be warned against suddenly drawing back from the needle as it penetrates the plexus. The solution is then slowly injected. With the index fingers of the right and left hands the sternal and acromial ends of the clavicle are determined. The thumb is placed in the midpoint of these limits and an iodine mark is made there. This point is just external to the external jugular vein. Medially to the iodine mark the fingers press gently into the tissues over and close to the clavicle, and thus determine the pulsations of the subclavian artery. The first rib is now located by pressing the fingers into the supraclavicular fossa; this becomes increasingly difficult with the fatness of the patient. With the type of needle already described a wheal is made over the spot through which the needle is to penetrate. The needle, never with the syringe attached, is then forced through the skin and fascia of the neck, great care being taken to avoid the external jugular vein. The cheek blowing manœuvre already mentioned can be used here to considerable advantage. With care the needle is pushed into the skin for ½ to 1 cm. in the general direction already described. As soon as the characteristic tingling is felt by the patient the needle is held stationary, the syringe attached to it, and the novocain slowly injected. If one has successfully impinged upon the cord of the plexus a sense of resistance

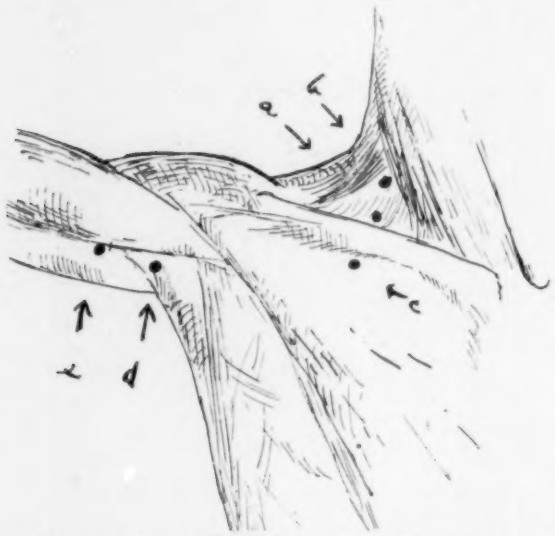


FIG. 3.—Points of entry for needles in different routes of plexus anæsthesia. a, Kulenkampff. b, Mulley. c, Babitzky-Hohmeier. d, Hirschel. e, Capelle.

is felt during the injection of the solution, which differs markedly from that felt when the needle is in loose subcutaneous tissue. The paræsthesias also increase momentarily until the action of the novocain causes its disappearance. The median nerve is usually first touched with the needle as it is most superficial, and then the radial which is behind it. One can now continue to inject the rest of the plexus. It is very helpful, if from time to time one lets the needle lie loose in the tissue and to observe if it is moved by the pulsation of the underlying subclavian artery. This manœuvre confirms the correctness of the needle's direction, and also warns us of the proximity of the large vessel. If the needle strikes the first rib without having produced the characteristic paræsthesias in the finger tips, it is certain that it has not been introduced in the proper direction; under such conditions the needle is withdrawn and re-inserted with its direction somewhat varied. The plane in which the needle enters the tissues should be at right angles to the trunks of the plexus, as in this way the chances of striking them are at their best. If one has directed the needle too far medially, then the subclavian artery is likely to be penetrated; if this occurs a definite sense of resistance is felt, and bright red blood will flow through the needle. This emergency is met by the immediate withdrawal of the needle 2 to 3 mm. to bring its point out of the lumen of the vessel and with the injecting of a little of the novocain-adrenalin solution into the tissues about the artery, which hastens the closure of the vessel. At times a little pressure with a piece of gauze over the artery is used in addition. We have never seen any harm result from this type of vessel injury. The omo-hyoid muscle, as it lies over the nerve trunks, can occasionally prevent a successful plexus block; in such a case, by a slight motion of the shoulder, the muscle can be brought into relief and the needle passed under it.

FIG. 4.—Plexus anæsthesia in sitting posture. The direction of the entering needle is well shown.

DURATION OF THE ANÆSTHESIA AND STRENGTH OF SOLUTION

In general, the anæsthesia lasts from two to three hours, the strength of the solution having a determining effect on this. Our first work in plexus anæsthesia was with 25 c.c. of a 2 per cent. solution* (10 c.c. in children). Then for a long time we used 12½ c.c. of a 4 per cent. solution which proved very satisfactory and gave anæsthesia lasting from three to four hours. At the present time 25 c.c. of a 2 per cent. solution is most frequently used. It has been found that the greater the degree of shock and the greater the

* Novocain solution to which is added 2 drops of adrenalin to each ounce.

loss of blood, the weaker the solution that could be used with satisfactory results. One must not expect to compensate a poor technique in injecting the plexus with the use of a stronger solution than usual. Basically, the duration and effectiveness of the anæsthesia depends on the exactness with which the plexus has been blocked. One can differentiate three degrees of anæsthesia: (1) The deepest type leads to complete loss in the use of the arm; unless the patient assures himself of the fact by visual evidence, he is unaware of possessing an upper extremity; motor, sensory and kinæsthetic senses are lost; this type of anæsthesia is ideal and therefore only infrequently obtained; (2) in this degree of anæsthesia one obtains a deep motor paralysis with insensibility to pain but without complete loss of kinæsthetic senses; (3) in this degree of anæsthesia the injection has been but indifferently done, but one still gets a sufficient degree of anæsthesia to permit of some operative procedures, provided the nerves included in the block are the ones supplying sensation to the region involved in the operation. Effectiveness of the anæsthesia is evidenced by a heaviness of the arm and forearm with a decrease in any previous pain. Ataxic movements of the arm when the patient attempts purposeful movements, is another manifestation of the anæsthesia. The supraclavicular nerves which to some degree supply the upper arm with sensation are completely eliminated, only, if at the same time that plexus block is induced, these nerves too are anæsthetized, by injecting them at the posterior border of the sternocleidomastoid muscle.

FIG. 5.—Plexus anæsthesia with patient in recumbent position.

UNTOWARD EFFECTS AND DANGERS

Because of a possible paralysis of the large nerve trunks incident to this type of anæsthesia, vasomotor reactions lead to a plethora of arm, forearm and hand. It does not as a rule lead to any disturbing features. The explanation of this phenomenon is probably as follows: the novocain solution is injected in such proximity to the arteries that it has a paralyzing effect on the small nerves penetrating its arterial coats, with a consequent dilatation of the artery and an increased blood supply to the parts. The sympathetic fibres on that side of the neck receiving the injection are occasionally involved; this manifests itself clinically by a hyperæmia and perspiration on that side of the face. Partial and temporary paralysis of the phrenic nerve is another possibility which manifests itself by weakened movements of the diaphragm on the affected side. Haertel and Keppler[9] by fluoroscope

control have demonstrated this condition. Fear of injury to the apex of the lung is not without its foundation. Capelle [8] reports one of the first of such occurrences—an emphysema of the lungs, following penetration of the pleura and lung substance by the needle. Further such occurrences have been reported by Brunner,[10] Klauser,[11] and Siebert.[12] Kulenkampff has reported on the train of symptoms attendant on such an accident: dyspnœa, chest pain, sense of chest oppression and sometimes slight cyanosis. Permanent or partial damage to the plexus has also been reported by Borcher,[13] Haertel,[14] Hirschler,[15] Keppler,[16] Flesch - Thebesius.[17] Haertel himself, however, considers the cause of this trouble not yet clearly defined. In this clinic we have never seen any serious damage to the plexus resulting from this type of local anæsthesia. We have on occasions seen irritation of the plexus due to the needle passing beneath the first rib and coming into contact with the pleura. At no time has damage to the lung been observed. One should be particularly careful of this complication in the emphysematous patient because in him the apex of the lung is, because of its distention, in nearer apposition to the plexus than is normal. Another thought to have in mind with these patients is that the inflated apex may also raise the level of the clavicle and in this manner cause the needle to take a false direction in seeking the plexus. Paralysis of the arm following a brachial block has never been met with in our experience. The involvement of the sympathetic and phrenic nerves have already been discussed and are without significance. Not infrequently patients will show a marked reaction following the injection of the solution, especially when the injection is made about the head or neck. The patient will show palpitation, marked pallor and restlessness. We believe this reaction to be due, not to the novocain, but to the adrenalin. The symptoms are similar to those seen when adrenalin is given to a patient for an acute attack of asthma. While the omission of adrenalin slows the effectiveness of the anæsthetic, it dispels the fear and anxiety which this reaction arouses in some patients. At this hospital, 1000 brachial plexus anæsthesias have been given since 1911. It is felt that this is a fairly large number on which to base an opinion as to the effectiveness of this procedure.

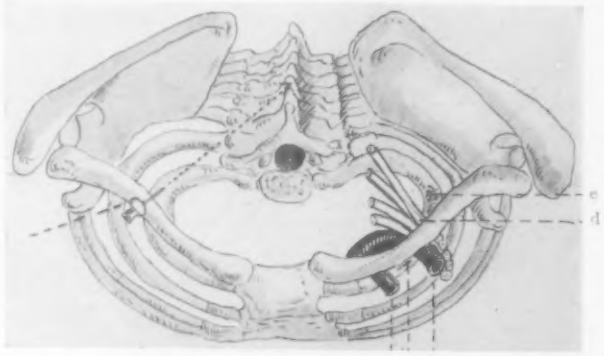


FIG. 6.—The plexus and related structures as seen from in front and above. Compare with Figure 1. Note well the direction of the entering needle, which, if long enough would strike the tip of the 2nd and 3d dorsal vertebræ.

SUMMARY AND CONCLUSIONS

The supraclavicular method of plexus block is teachable and learnable. It eliminates pain from the arm, forearm and hand, and produces a motor

and sensory paralysis directly proportionate to the degree of skill with which the anæsthesia has been produced. One can inject the nerve trunks successfully, provided their location external to the subclavian artery and its relation to the other anatomic points is carefully visualized. The first rib marks the deepest point of needle penetration. The direction of the entering needle should be such as if one wished to strike the spinous process of the second or third dorsal vertebra. After striking the median nerve (the desideratum in plexus anæsthesia) as manifested by tingling and various paræsthesias in the finger tips, 25 c.c. of a 2 per cent. solution of novocain-adrenalin are slowly injected. Within ten to twenty minutes anæsthesia is complete. If one is working high up on the shoulder or in the region of the axilla, the intercosto-humeral nerve, a branch of the second intercostal, must be blocked. In careful hands plexus anæsthesia is practically without danger. Care must be taken to avoid injury to the apex of the lung, the external jugular vein, and subclavian artery. To avoid injury to the cords of the plexus a very fine calibre needle is to be used. Puncture of the subclavian artery, while not a desirable accident, should cause no fear; the immediate withdrawal of the needle into the surrounding tissue with the injection of the novocain-adrenalin solution will hasten the closure of the artery; the needle is then withdrawn and compression with a piece of gauze maintained for a few moments. Occasionally there is paralysis of the phrenic nerve or sympathetic of the neck; both conditions are temporary and without serious consequences. The duration of the anæsthesia is from two to four hours. In many cases this anæsthesia can be used for the inspection of painful injuries preliminary to any operative work. Plexus anæsthesia is practical, has well defined usefulness, and can be utilized even in cases where a general anæsthetic would but add to an already existing shock.

BIBLIOGRAPHY

[1] Gray's Anatomy.

[2] Heinrich Braun: Oertliche Betäubing.

[3] Hirschel: Die Anaesthesierung des Plexus brachialis in der Achselhöh le bei operativen Eingriffen an der oberen Extremität. Verh. d. D. Ges. f. Chir. 1912. I. S. 348. und M. med. Wochenschr., 1912, No. 22.

[4] Perthes: Ueber Leitungsanaesthesie mit Zuhilfenahme elektrischer Reizung. M. med. Wochenschr., 1912, No. 47.

[5] Mulley: Eine Modification der Plexusanaesthesie nach Kulenkampff behufs Vermeidung einer Pleuraverletzung. Beitr. z. klin. Chir., 1919, Bd. civ, S. 666.

[6] Hohmeier: Die Lokalanaesthesie der Extremitäten. D. Chir. Kongr., 1914.

[7] Babitzky: Neuer Weg zum Plexus brachialis zwecks Anaesthesierung. Zentr. f. Chir., 1918, No. 13.

[8] Capelle: Die Anaesthesie des Plexus brachialis, ihre Gefahren und deren Vermeidung. Beitr. z. klin. Chir. 1917. Bd. civ, S. 122.

[9] Härtel u. Keppler: Erfahrungen über die Kulenkampff'sche Anaesthesie des Plexus brachialis. Arch. f. klin. Chir. Bd. ciii, H. 1.

[10] Brunner: Zur Frage der Phrenicuslähmung nach Plexusanaesthesie. Zentr. f. Chir., 1923, S. 1104.

[11] Klauser: Phrenicuslähmung bei Plexusanaesthesie. Zentr. f. Chir., 1913, vol. x, p. 599.

[12] Siebert: Ueber einige neuere Anaesthesierungsverfahren mit besonderer Berucksichtigung der Plexusanæsthesie. Med. Klinik, 1912, No. 48.
[13] Borchers: Die supraclaviculäre Anaesthesierung des Plexus brachialis. Zentr. f. Chir., 1912, No. 26.
[14] Härtel: Die Lokalanaesthesie. Neue Deutsche Chirurgie, Enke, 1920.
[15] Hirschler: Nervenschädigungen bei Plexusanaesthesie. Zentr. f. Chir., 1913, S. 766.
[16] Keppler: Die Anaesthesierung der unteren Extremität mittelst Injektion auf die grossen Nervenstämme. Arch. f. klin. Chir., 1913, Bd. c.
[17] Flesch-Thebesius: Länger dauernde Armlähmung nach Kulenkampff'sche Plexusanaesthesie. Zentr. f. Chir., 1919, No. 33.

TRAUMATIC OSTEOPOROSIS OF THE CARPAL BONES*

By Joseph Buchman, M.D.

of Brooklyn, N. Y.

FROM THE SERVICES OF DR. ROYAL WHITMAN, HOSPITAL FOR THE RUPTURED AND CRIPPLED, NEW YORK; AND DR. SAMUEL KLEINBERG, UNITED ISREAL-ZION HOSPITAL, BROOKLYN, NEW YORK

A considerable volume of literature has been written and many hypotheses have been advanced to explain a rare condition most commonly called Kienböck's disease of the carpal semilunar and Preiser's disease of the carpal scaphoid. These authors describe what I believe to be different manifestations of a similar process, for which I will submit the term "Traumatic Osteoporosis of the Carpal Bones" as more appropriate. No one has as yet presented an etiologic, pathogenic, or pathologic picture that has met with general acceptance. We know that the condition exists, and that it is a distinct entity, but further than that there is no consensus of opinion. With the hope to clarify the subject, I desire to report several cases and review the literature.

CASE REPORTS

Case I.—T. N., a housewife of twenty-four years of age, was seen by me for the first time in the Out-patient Department of the Hospital for the Ruptured and Crippled, on the service of Dr. Royal Whitman, during the latter part of August, 1926. She complained of pain in the right wrist. About three years previously, the patient fell on her right hand, forcing it into extreme hyperextension. Her physician told her that she had a fracture of one of the small bones of the wrist. She was treated with a plaster-of-Paris bandage, which held the wrist in extension for four weeks. After that she found that she could not hyperextend her wrist, and that supination of the forearm was limited. Since that time, climatic changes have frequently caused the patient to become conscious of her wrist because of stiffness and pain. On August 14, 1926, she was awakened by severe pain in the wrist, which disabled her considerably, and which has disturbed her sleep ever since. There was no history of any other injury save that described above.

Examination on admission showed that the wrist was greatly guarded. All extremes of motion were limited by severe pain. There was no swelling, redness, or increased heat. Tenderness was very marked over the scaphoid bone. X-ray examination (Fig. 1) showed a solution of continuity through the middle of the scaphoid. The bone adjoining the line of fracture presented areas of absorption and areas of increased calcification. The lateral view presented the same mottled appearance.

A circular plaster-of-Paris bandage was applied to hold the hand in hyperextension, and it was retained for three weeks. During this period, there was only slight relief from pain. On removal of the plaster bandage, no improvement was found. All motions were greatly limited and the pain had increased, especially at night.

The patient was, therefore, admitted to the hospital, and on September 27, 1926, I removed the scaphoid through a dorsal incision. The patient made an uneventful recovery, and nine days later she was discharged wearing a duraluminum cock-up splint holding the wrist in hyperextension. Physiotherapy was instituted and continued for five

* Read before the Orthopædic Section of the New York Academy of Medicine, December 16, 1927.

weeks. Thereafter she had no active treatment. On August 27, 1927, there was a complete range of motion in the wrist and the grasp was fully as strong as that of the left hand. Röntgenograms showed that the margins of the os magnum and semilunar adjacent to the former site of the scaphoid were somewhat roughened. The patient was symptomless except on changes of weather, when she at times became conscious of her wrist.

At the time of the operation, the soft tissues were normal, and there was no evidence of any pathology in the joint, in the adjoining bones, nor in their cartilaginous coverings. The scaphoid consisted of two fragments united by fibrous tissue. The cartilaginous coverings were normal in appearance and texture. The bone was found to be very fragile. Along the line of fracture, there were very small cysts lined with shiny membranes.

FIG. 1.—Case I. Antero-posterior view showing: *a*. Solution of continuity through the middle of the scaphoid. *b*. Areas of absorption. *c*. Areas of eburnation.

Microscopic examination (Fig. 2) revealed that near the line of fracture the marrow was displaced by areas of loose and dense fibrous tissue. The bony structures were thinned and invaded by fibrous tissue. Whatever bone remained was normal, in that there was no diminution nor increase of calcification. There was no osteoid tissue present. There was no evidence of necrosis, of round-cell infiltration, nor of giant cells. The sections unfortunately did not include any of the cysts noted macroscopically. There was a definite increase in the number and size of the blood-vessels. The farther distant from the line of fracture, the less abnormal the tissues were found to be. The cartilage was not involved.

In this case there was a distinct history of trauma; and the pathological findings in the scaphoid consisted mainly of thinning of bone, that is, osteoporosis; replacement of bone and marrow by fibrous tissue; and an increase in the number and size of blood-vessels.

CASE II.—I. H., a taxi-cab driver of thirty years of age, was seen by me for the first time September 5, 1926, in the Out-patient Department of the Hospital for the Ruptured and Crippled, on the service of Dr. Royal Whitman. He stated that five days previously, an automobile in which he was riding was struck in the rear, and he

was thrown forward so that his wrist became hyperflexed and the dorsum of the hand impinged against the corner of the car. There was immediate pain and disability which prevented him from performing his duties as a chauffeur. There was no history of any previous injury, pain, nor discomfort.

Examination showed a considerable degree of swelling about the wrist. There was tenderness over the scaphoid bone, but none in the anatomical snuff box. There was no limitation of motion. There was pain on hyperextension and at the extremes of all the other motions, especially radial deviation.

FIG. 2.—Case I. High power magnification of scaphoid showing: *a*. Loose fibrous tissue. *b*. Dense fibrous tissue. *c*. Bony structure invaded by fibrous tissue. *d*. Normal bone.

X-ray examination (Fig. 3) showed a fracture through the middle of the scaphoid. The line of fracture was smooth and apparently was not of recent origin. The lowermost portion of the scaphoid was sclerosed and the area adjoining the line of division presented small areas of absorption. The lateral view presented the same mottled appearance.

Immobilization of the wrist with adhesive plaster for three weeks gave no relief. At his urgent request for operation, the patient was admitted to the hospital on October 19, 1926, because of the increasing pain, disability, and inability to sleep. On the following day, I removed the offending bone through a dorsal incision. The patient made an uneventful recovery, discharged six days later, with plaster-of-Paris molded cock-up splint.

Examination one month later showed that the hand was held in slight radial deviation. The grasping power had increased markedly, and the patient stated that there was comparatively little pain. There was 30 degrees dorsal flexion, 30 degrees palmar flexion, 20 degrees radial deviation, and 10 degrees ulnar deviation. I did not see the patient again, but in January, 1927, I was informed that he had returned to his previous occupation of taxi-cab driver.

At the time of operation no evidence of recent fracture was perceptible. The soft tissues, the joint space, and the adjoining bones were normal. The scaphoid was divided into two parts, which were united by connective tissue. The cartilage appeared normal. The small cysts, noted in the first case, were not seen in this specimen. Microscopic examination (Fig. 4) revealed a picture identical with that of the first case.

This case is the only one of my series in which the alleged injury was apparently not the cause of the lesion found at operation. Here, too, the

pathological findings were osteoporosis, increased blood supply, and fibrous replacement of bone and marrow.

CASE III.—M. T., a housewife of thirty-four years of age, was admitted to the Out-patient Department of the Hospital for the Ruptured and Crippled, on the service of Dr. Royal Whitman, April 7, 1927, stating that, seven months previous, she had fallen while pole vaulting. She landed on the palm of her hand, forcing it into hyperextension. Immediately thereafter, she had pain in the wrist and discomfort. She had no medical care, but continued to discharge her household duties. This went on until four months ago, when she fell again and the pain became much more severe. Her pain was greatest on lifting heavy weights.

FIG. 3.—Case II. Antero-posterior view showing: *a*. Fracture through the middle of the scaphoid. *b*. Sclerosis of inferior portion of the scaphoid. *c*. Areas of absorption.

Examination showed no swelling, nor redness over the wrist. No abnormalities were palpable. There was tenderness at the base of the first metacarpal bone, but none in the anatomical snuff box. Traction on the first and second fingers did not cause any pain. Pressure applied along the course of the first and second fingers caused pain, but to a lesser degree in the latter. This pain was referred to the base of the first metacarpal bone. Hyperextension and flexion, though slightly limited, were accompanied by pain. The lateral motions were not restricted. Stereoscopic X-ray examination (Fig. 5) showed that the scaphoid presented three very small distinct vacuoles, all arranged in one line through the centre of the scaphoid, and a very fine line of fracture running through these vacuoles. There was no displacement of the fragments. The semilunar showed slight vacuolation. The stereoscopic lateral view showed a fracture through the middle of the scaphoid with areas of rarefaction.

Conservative treatment was instituted in the form of a circular plaster-of-Paris bandage for the forearm, wrist and metacarpus, holding the hand in hyperextension. This was removed three weeks later, and the patient was found to be relieved of her symptoms. Objectively the wrist was negative.

Eight months later the patient returned, stating that her relief was only temporary.

Her pain was now sufficiently severe to cause her to plead for operative interference. Examination showed a somewhat swollen wrist, held in 30 degrees ulnar deviation. Hyperextension, hyperflexion, and radial deviation were limited and painful. Pressure along the course of the third finger and in the anatomical snuff box caused pain in the scaphoid. X-ray examination (Fig. 6) revealed that the osteoporosis, previously seen in the semilunar, had cleared up. The scaphoid, however, showed extension of the pathological process, for the three vacuoles originally present were now fused into one definitely enlarged area of rarefaction, while the line of fracture through the middle of the bone was more marked.

FIG. 4.—Case II. High power magnification of scaphoid showing: *a*. Loose fibrous tissue. *b*. Dense fibrous tissue. *c* Increase of blood-vessels. *d*. Normal bony trabecula.

This again is a case in which there was an injury to the wrist, resulting in a pathologic change similar to the first two cases. Here the semilunar as well as the scaphoid was involved.

CASE IV.—J. D., a nineteen-year-old boy, was seen by me for the first time on May 11, 1927, in the Out-patient Department of the United Israel-Zion Hospital, service of Dr. Samuel Kleinberg. He stated that a few days previously he fell off the running board of an automobile and landed on his hyperextended hand. Pain had been present ever since. One year ago he fell and injured the same wrist. At that time he had no medical care. Pain had persisted for one month during which he wore a wrist band. Since that time he has had no discomfort until the recent accident.

Examination showed a slightly swollen wrist. There was slight tenderness over the scaphoid, both in its dorsal and palmar aspects, and in the anatomical snuff box. Percussion over the second knuckle caused pain in the region of the scaphoid. All extremes of motion were painful, especially palmar flexion and ulnar deviation. X-ray examination revealed a fracture through the middle of the scaphoid with no displacement of the fragments. There was a circular area of rarefaction near the margin adjacent to the os magnum. Mottling was present along the line of fracture and a heaping up of bone was seen over the outer surface.

The lesion in this case was believed to be similar to that in the previous cases, that is, rarefaction of bone resulting from an injury, and the diagnosis made was traumatic osteoporosis with fracture of the scaphoid.

A circular plaster-of-Paris bandage, holding the hand in hyperextension, was applied. Three weeks later, the support was removed. After two weeks of physiotherapy, the patient was relieved of all symptoms.

On August 17, 1927, the patient was recalled. He reported complete relief from all symptoms and disabilities. Radial deviation was limited to 10 degrees (opposite side 30 degrees). There was slight crepitation over the scaphoid on flexion and extension of the wrist. The anatomical snuff box was fuller than that on the opposite side. X-ray examination revealed that the osteoporosis seen previously had in part disappeared. The ulnar aspect of the scaphoid presented a heaping up (callus formation) which explains the fulness of the anatomical snuff box.

FIG. 5.—Case III. Antero-posterior view showing: *a*. Three small vacuoles running through the thickness of the scaphoid with a fine line of fracture through them. *b*. Slight vacuolation of the semilunar.

CASE V.—F. S., a twenty-nine-year-old man, was seen for the first time on September 30, 1926, in the Out-patient Department of the Hospital for the Ruptured and Crippled, service of Dr. Royal Whitman. He stated that two years previously he fell from a cliff forty feet high, injuring his right wrist and right ankle. The patient had conservative treatment for his wrist in the form of closed manipulation, plaster-of-Paris support, and physiotherapy. He complained of inability to do heavy work, pain on exertion, partial loss of sensitiveness in his fingers, and a tendency to become easily fatigued.

Examination revealed a thickening of the carpus, most prominent on the dorsum. There was a loss of about one-third of the normal range of flexion and extension. Lateral deviation, pronation, and supination were normal. Motions in the wrist were accompanied by crepitation. There was considerable laxity in the radio-carpal joint. The grasping power was definitely diminished, and the forearm was atrophied one-half inch.

Stereoscopic X-ray examination showed in the antero-posterior views that the os magnum, the unciform, the cuneiform, and the second, third, and fourth metacarpal bones were all displaced dorsally. The scaphoid appeared to be fractured, and there was increased density in the line of fracture. In the semilunar, there were two small, punched-out, circular areas. The os magnum also presented areas of rarefaction. The pisiform appeared to be in alignment with the scaphoid and the semilunar bones, which,

in turn, were in normal relationship to the radius. Stereoscopic X-ray of the lateral view showed that the outlines of all of the carpal bones were somewhat irregular. There was mottling in all of the bones, especially in the os magnum, scaphoid and semilunar. The semilunar bone, scaphoid, trapezium, and trapezoid appeared to be in their normal line with the radius. The os magnum, cuneiform, and unciform were displaced posteriorly. The second, third, and fourth metacarpal bones were in proper alignment with the os magnum and the unciform.

FIG. 6.—Case III. Antero-posterior view taken eight months later showing: *a*. Fusion of vacuoles shown in Fig. 5 and enlargement of the area of rarefaction. *b*. Diminution of vacuolation in the semilunar.

This patient had a mid-carpal dislocation and a fracture of the scaphoid, with traumatic osteoporosis of all the carpal bones, especially the scaphoid, semilunar, and os magnum. Conservative treatment was advised. The patient was then lost sight of.

CASE VI.—J. F., a thirty-year-old-man, was admitted to the Out-patient Department of the Hospital for the Ruptured and Crippled, service of Dr. Royal Whitman, on November 26, 1927. He gave a history of pain in the left wrist for the past five months. The onset was gradual, and the symptoms recurred from time to time. Pain was only present on hyperextension and hyperflexion. There was no recollection of any injury to the wrist. The patient's work consisted of pushing and pulling a truck.

Examination showed a slightly thickened wrist, most marked in the region of the semilunar bone. Pressure in this region caused pain. The motions of the wrist were unrestricted. Extreme hyperextension, hyperflexion, and ulnar deviation caused pain. There was no loss in the strength of the hand grasp.

X-ray examination of the antero-posterior view (Fig. 7) showed that the semilunar bone was thinned. The proximal portion was irregular and presented areas of rarefaction and sclerosis, while the distal portion showed increased calcification. The os magnum and unciform revealed circular areas of halistersis. The articulating surface of the radius was irregular and thickened. The lateral view (Fig. 8) showed that the semilunar was thinned and widened in its dorso-palmar axis, and its upper surface was irregular. The semilunar, as well as the os magnum, presented areas of rarefaction and sclerosis, while the joint space between the radius and the carpus was considerably diminished.

Though there was no distinct history of trauma, the possibility of repeated minute traumata incidental to pushing and pulling a truck is very likely. The röntgenographic findings are those of osteoporosis of the semilunar, the os magnum, and the unciform, with secondary osteoarthritic changes involving the radiocarpal joint. Hence, we believe this instance to be similar to the previous cases, and we diagnosed it as traumatic osteoporosis of the semilunar, the os magnum, and the unciform, with secondary osteoarthritic changes in the wrist.

Because of the comparatively slight discomfort the patient experienced, it was deemed advisable to institute physiotherapy for the present. We cannot as yet report the result obtained because of the brevity of the period the patient has been under observation.

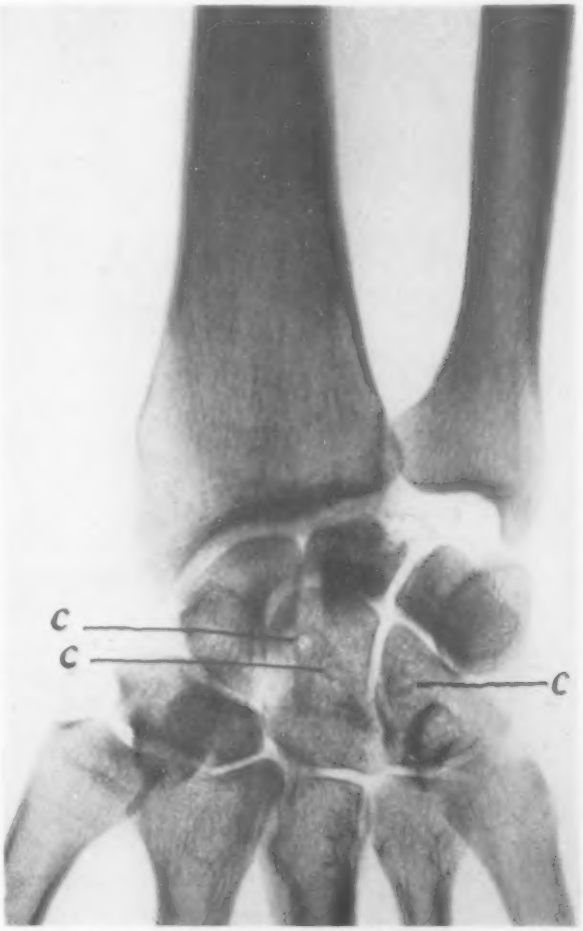


FIG. 7.—Case VI. Antero-posterior view showing: *a*. Irregularity of the upper margin of the semilunar bone. *b*. Areas of rarefaction and sclerosis of semilunar bone. *c*. Circular areas of halistersis in the os magnum and unciform.

CASE VII.—M. K., a thirty-two-year-old man, came on December 10, 1927, to the Out-patient Department of the Hospital for the Ruptured and Crippled, service of Dr. Royal Whitman, stating that he has had pain in his left wrist on and off for the past two years. At no time was there a gross injury that the patient can recall. His work was that of a cutter, and with the palm of his left hand he pressed down the materials cut. Examination revealed no swelling nor limitation of motions. Extreme flexion and extension were painful. There was tenderness proximal to the third metacarpal bone. X-ray examination (Fig. 9) showed that both the os magnum and the unciform presented several small round areas of rarefaction.

This case, like the former, gave a history of repeated minute traumata incidental to his work, and a röntgenographic appearance of osteoporosis.

My Cases V, VI, and VII are, to my knowledge, the only ones recorded in the literature in which there is an osteoporosis of the os magnum and unciform resulting from injury. Destot alone, without citing any definite instance, mentions the possibility of such a condition occurring in the os magnum.

Summary of Case Reports.—Seven cases are reported. In three of these the scaphoid was affected. In the fourth case both the scaphoid and the semilunar were involved. The fifth case presented osteoporosis of the semilunar, scaphoid, and os magnum. The sixth case showed osteoporosis of the semilunar, os magnum, and the unciform. In the seventh case the os magnum and the unciform were similarly affected. All of these cases, with the exception of the second, sixth, and seventh, gave a definite history of injury. In the second case, the trauma preceded the onset of symptoms, but the pathologic appearance leads me to believe that the process was present previous to the alleged injury. In the sixth and seventh cases, the nature of the patient's work make the likelihood of repeated traumata very real. Only four cases were treated, two conservatively and two by the operative removal of the offending bones. Of these, one of the non-operated cases is doing very poorly, while all of the others have good functional results. Thus, trauma is a factor in all of my cases and rarefaction or osteoporosis is the outstanding pathologic state. Hence, I believe the term traumatic osteoporosis is the most appropriate one for the condition described.

FIG. 8.—Case VI. Lateral view showing: *a*. Thinning, widening, and irregularity of the semilunar. *b*. Osteoarthritic changes in the radio-carpal joint.

Historical Data.—Numerous writers discussed this subject prior to 1910. Among these were Peste, Pfitzner, Wolf, de Quervain, Grosz, Witteck, Lilienfeld, Ehermeyer, and Natvig. Wolf expressed the opinion in 1903 that this condition was a compression fracture. Blau, in 1904, was the first one to note central halistersis or rarefaction in the scaphoid. In 1910, Georg Preiser, with the aid of röntgenological studies, describing five cases of involvement of the scaphoid; and Kienböck, somewhat later in the same year, collected twenty and added sixteen of his own cases of semilunar bone involvement.

Etiology.—This affection is found more commonly in the semilunar than in the scaphoid. Its onset may occur in one of three ways. First, there may be a definite injury, more commonly to the dorsiflexed hand, and occasionally to the palmar flexed hand. Second, there may be an insignificant

injury or repeated traumata. Third, there may be no history of injury at all. As an example of the latter, Mouchet and Pichat reported a case of semilunar disease in which the onset was characterized by occasional pains for several months, followed by a severe injury. Similarly, in my sixth case, there was no history of a definite injury; and its onset was characterized by occasional pains for five months.

The etiology of cases of scaphoid disease is not well known because of the few instances reported. In these there is usually a definite injury. Occasionally there is no knowledge of any trauma. All of my cases give a definite history of injury, except Case II, in which the X-ray taken five days after the trauma showed a long-standing condition.

Incidence.—Semilunar bone involvement is found, as shown in Table I, about four times more frequently in men than in women, and about four

TABLE I.

Author	Number of cases	Males	Females	Semilunars right	Semilunars left	Age incidence
Sonntag	67	45	22	..	..	Most of these between 17 and 35 years.
Kienböck	36	31	5	24	12	Twenty-three between 17 and 39 years.
Becker	20	..	..	16	4	Eight under 30, and 8 between 30 and 40 years.
Saupe	79	69	10	54	22	Fifty-four between 20 and 29 years.
Totals	202	145	37	94	38	

times more often on the right than on the left side. It most commonly occurs in the third and fourth decades of life. Goldsmith states that it is seven times more frequent in males than in females, two and a half times more frequent on the right than on the left side, and is usually in the third and fourth decades of life.

The cases of scaphoid involvement are most commonly men. The age period is about the same as in the semilunar cases. Here, too, the right is usually the affected side. In both types the lesion is mainly in manual workers. In my series of five cases of scaphoid disease, there were three males and two females, the right side was involved four times and the left side once, and the age incidence was between nineteen and thirty-four.

Course and Symptomatology.—The course of the disease as it exists in the semilunar bone divides itself into several typical stages. First, the acute stage which sets in immediately after the injury and lasts from a few hours up to several weeks. The injury is usually mild and the patient is not disabled. It is commonly of the nature of a sprain and is passed off as such. There is a mild grade of swelling, pain, tenderness over the semilunar, and limitation of extremes of motion, especially dorsiflexion, and, to a lesser degree, palmar flexion. This is followed by an interval of freedom from symptoms which may last weeks, months, or several years. The period of actual disease then sets in. This is characterized by progressively

increasing disability, swelling and tenderness over the semilunar bone. At first the pain is only present on extreme exertion; but gradually it becomes more and more persistent and annoying in character, until finally it is disabling. At this time and, to a less extent, when the symptoms are less severe, Finsterer's pathognomonic sign appears, namely, that, whereas normally the third knuckle is most prominent, in this condition it loses its prominence and may even recede because of the vertical shortening of the affected bone. The same author found that percussion over the third metacarpal bone caused pain in the semilunar. Pulling actions with the hand are painless, but when pushing is attempted, pain is experienced. The swelling, which is due to the hemorrhage resulting from the original trauma, and which sets in immediately after the injury, subsides after a while. This is followed by a slight permanent periarticular swelling which is caused by the repeated traumata to which the wrist is subjected when in use. Guye found that the carpus is somewhat shortened, and he has found a filling up of the normal depression that is present just distal to the radius. Kienböck states that, at times, on acutely flexing or hyperextending the wrist, there is a tender prominence over the dorsum of the semilunar. This swelling is due to the antero-posterior widening of the bone. Just distal to this swelling is a depression in which fragments and crepitation may be felt.

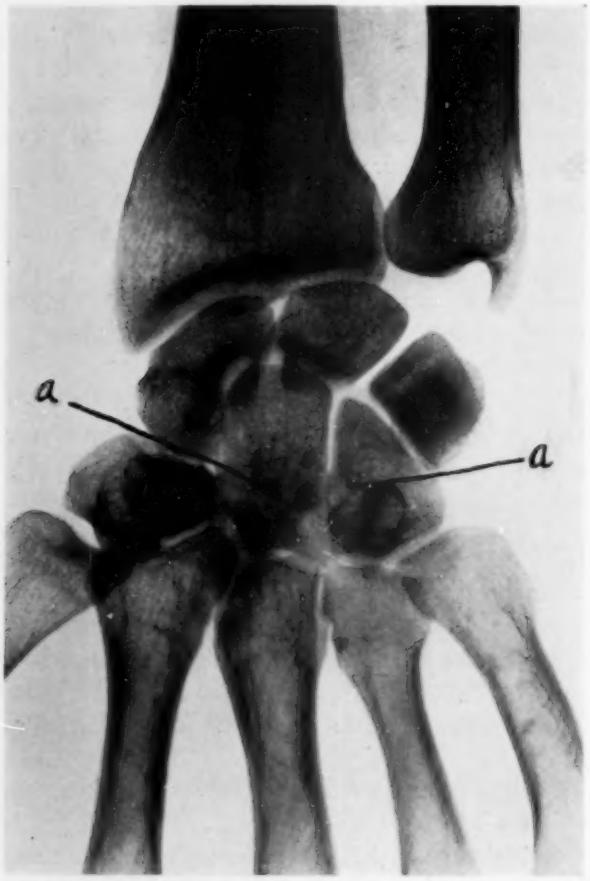


FIG. 9.—Case VII. Antero-posterior view showing small areas of rarefaction in the os magnum and unciform.

The course of the disease in the scaphoid is practically parallel to that of the semilunar. There may or may not be an original severe injury. More commonly, it is a slight trauma. The pain lasts for a comparatively short period of time, and it subsides for a varying interval. The quiescent stage may be characterized by a slight consciousness on the part of the patient that

something is wrong with the wrist. The active stage sets in gradually. The pain, at first slight and only felt on exertion, becomes more and more severe, and is finally present on rest. It may become very severe and excruciating, so that the sufferer may be unable to sleep, as is illustrated by my first two cases. The limitation of motion, which is at first active, finally becomes passive. Dorsiflexion is the most often restricted, while supination and pronation are usually free. The latter motions, however, were restricted in my first case. Objectively there is a slight swelling and tenderness over the scaphoid. Houget and Mouchet found that in their case the anatomical snuff box was obliterated. I found a similar phenomenon in my fourth case. This case also presented crepitation in the region of the scaphoid. My third case complained of pain in applying pressure along the course of the thumb and index finger, although the pain was less in the latter. The pain was referred to the base of the first metacarpal. In my fourth case, percussion over the second knuckle caused similar pain.

FIG. 10.—Case VIII. Antero-posterior view showing ulnar displacement of the carpus with the semilunar bone wedged between the styloid process of the radius and the scaphoid.

Röntgenography.—In the great majority of semilunar cases the X-rays are at first apparently negative. At a later date, the semilunar presents areas of rarefaction and areas of more intense calcification. The entire bone is darker than the other bones. The proximal portion is first involved. The bone becomes thinned in its vertical or short axis and widened in the

antero-posterior axis. As the disease progresses, the proximal portion may become fragmented and portions may disappear. The proximal convex surface becomes flattened and irregular, and the entire bone is thinned. In those cases which had a severe injury there may at times be a compression fracture. Other changes, secondary in nature, are of an osteoarthritic character. The head of the os magnum may be displaced upward. The articulating surfaces of the radius may show osteoporosis and the points of insertion of the ligaments may become roughened.

Most authors state that from the very beginning a very fine line of fracture may be noted on very careful study. The explanation for the apparent absence of line of fracture is thought by Gaza to lie in the fact that the fine lines can not be discerned in the lateral view because of the superimposition of bones, and in the antero-posterior views because of the thickness of the bone. Kienböck, while admitting that the fine lines of fracture may be noted from the very beginning, is inclined to believe that the fracture is secondary and pathological in nature.

Röntgen-ray studies of the scaphoid show that the characteristic appearance may be present as early as the second day after the injury, as shown by one of Preiser's cases. This, however, is unusual. First, there may appear one or more small circular or oval areas of halistersis or rarefaction in the centre of the bone. These soon fuse into one large area of lessened density. It is through this area that a fracture appears later. Constriction at the middle of the body occurs, and the bone presents darker and lighter areas, areas of absorption and eburnation. The entire bone is darker than the surrounding bones. In the late stages secondary osteoarthritic changes take place.

The interpretation of the X-ray picture of a supposed primary fracture has been much discussed by many authors. Preiser, like Kienböck, is of the opinion that there is no fracture at first, that it is only a later occurrence, and that it is pathological in nature. Haenich has been able to demonstrate that Preiser's original plates show fissure fractures. Hirsch agrees with the latter author, stating that fractures cannot always be demonstrated unless pictures are taken from many angles. As a further proof, he has found on operation, in one case one day after injury, and in another case three days after injury, fresh fractures running through the rarefied areas which contained fresh blood and bloody pulp in the body of the bone.

Pathogenesis and Pathology.—The pathogenesis of these conditions is unsettled. First, Preiser, in describing the entity as it exists in the scaphoid, and then Kienböck, in writing on the involvement of the semilunar bone, advanced the hypothesis of injury to blood-vessels incidental to the tearing of the dorsal and lateral ligaments. This occurs subsequent to a momentary dislocation of the semilunar or the scaphoid bone, as the case may be. These authors base their conclusions on Von Lexer's anatomical works which show that these ligaments carry the main blood supply to the bones in question. As a result of the interfered circulation, rarefaction occurs, and the walls

become so thin that a pathological fracture is superimposed. If the secondary blood supply by way of the volar ligaments is sufficient, restitution takes place, as has been noted in several instances. This has been contradicted by the observations of a number of writers. Castruccio's experiments show that great forces are necessary to produce injury in the ligaments carrying the blood supply. Furthermore, operative findings do not substantiate this hypothesis. Lériche points out that rarefaction cannot take place without a hyperæmia. With an interfered circulation the density of the bone should remain in status quo. The inadequacy of this explanation is demonstrated by the following case which has come under my observation.

Case VIII.—A. H., a twenty-six-year-old chauffeur, was seen by me in the United Israel-Zion Hospital, service of Dr. Samuel Kleinberg. He complained of an injury to his right wrist. X-ray examination showed in the antero-posterior view (Fig. 10) that the carpus was displaced to the ulnar side. The semilunar bone was wedged in between the scaphoid and the styloid process of the radius. The space where the semilunar bone normally appears was vacant. The cuneiform was displaced upward, so that it impinged on the styloid process of the ulna. The lateral view showed the semilunar bone displaced slightly forward without rotation, its posterior cornu resting on the head of the os magnum. The scaphoid was slightly rotated so that the upper segment was more anterior than normal, while the cuneiform was displaced upward and posteriorly.

I manipulated the wrist and the dislocation was reduced. The patient made an uneventful recovery. Eight months later, X-ray examination showed a perfectly normal wrist. The patient had returned to his former work and was entirely symptomless.

One can hardly gainsay that, in this particular instance, there must have been an extensive tearing of the ligaments. Nevertheless, healing has occurred without any evidence of the disease under consideration.

Axhausen brings forth his theory of aseptic primary bone necrosis with a superimposed pathologic fracture to explain these conditions. He states that bland mycotic emboli lodge in these bones, that aseptic necrosis occurs, that a pathologic fracture then supervenes, and that a separation process and structural changes subsequently occur leading to deformity of the bones. He states that Aschoff has accepted this possibility and further supports his contentions by microscopic studies which show areas of aseptic bone necrosis. Kappis and others agree with him in part, stating that it is either an embolic or a traumatic necrosis. Though other observers have found areas of necrosis, some of them find no evidence of embolism. Some authors are of the opinion that the necrosis is secondary to a crushing of the spongiosa. Hirsche's operative findings of fresh blood detritus caused by a crushing fracture contradict Axhausen's theory. My own microscopic studies and those of other writers do not reveal any necrosis. Furthermore, it is difficult to explain just why bland embolism should occur in this select group of individuals, under these particular conditions, during this more or less limited age period, in the semilunar, scaphoid, and os magnum.

Miller believes that these conditions are the result of abnormal pressure due to anatomical anomalies. He has not shown the exact nature of these anomalies, nor has he shown why they should occur under the given con-

ditions in active individuals who present no evidences of other abnormalities.

Destot thinks that the condition is due to poor circulation of the bone that it is a degeneration, and that all cases are not fractures.

Fontaine adds the infectious theory on the basis of Mouchet and Lecene's findings of attenuated osteomyelitis. This is controverted by the practical absence of any report showing inflammatory reaction on microscopic examination.

Baum concludes that the regenerative ability of the wrist bones is defective and expresses itself in connective-tissue formation. According to this author, the pathogenic sequence is trauma resulting in fracture, followed by a partial healing with a pseudo-arthrosis and an incomplete callous formation. In this connection it is interesting to note some of the conclusions drawn by Johnson from his experimental study of the healing processes in injuries to the carpal scaphoid. He states: "(1) Bone repair takes place in the scaphoid in exactly the same manner as in the medulla of the diaphyses of long bones, but the process is not as active or extensive. (2) There is no interference with the vascularity of the fragments of a fractured scaphoid which would cause necrosis or interference with hyperæmia and secondary delayed union or non-union." In view of these conclusions, Baum's theory is untenable. Johnson's work also eliminates the theory that, as a result of a crushing of the spongiosa, there is an interference with blood supply and a subsequent rarefaction.

Wollenberg's case of disease of the scaphoid came to autopsy, and on examination he found necrosis, partly decalcified bone, other connective-tissue injury, periosteal callous in the region of the cortical fracture covered by periosteum, new bone formation and cysts in the line of fracture. His conclusions were: (1) that the condition consisted of an old fracture, or at least, an incomplete fracture; (2) that a cyst formation of the nature of a localized osteitis fibrosa developed in the region of the fracture, and (3) that the walls of the cysts contained many giant cells so that it became a question of giant-cell sarcoma. He felt justified in ruling out the latter condition.

My microscopic sections show fibrous tissue replacement of bone and marrow and increase in the blood-vessels. The bony structures are thinned. These observations coincide, as far as they go, with those of Wollenberg. The explanation for these changes may lie in Lériche's teachings, which are to the effect that there can be no rarefaction without a hyperæmia. He believes that all peripheral traumatisms are accompanied by a disturbance in the vasomotor equilibrium, more or less lasting, which starts with a constriction and ends with a dilatation. He calls this phenomenon the traumatic axone reflex (des reflexes d'axone traumatique). His pathogenic sequence in this condition is trauma, giving rise to a traumatic axone reflex, which, if persistent, will give rise to absorption of bone.

Of all the theories presented to explain the pathogenesis and pathology of this disease, none are, to my mind, as plausible as that of Lériche's traumatic axone reflex. This hypothesis satisfies all of the conditions pre-

sented clinically, röntgenographically, and pathologically. This concept explains the great variation in the degree of trauma noted in the various cases, for an injury, minute or gross, is capable of starting the process. According to this belief, primary fracture, though most common, is not essential. Absorption begins in the presence or absence of fracture, and because of it fracture and extensive destruction occur secondarily. The reasons for the gradual increased symptoms and the gradual clearing up of the condition in those cases that yield to conservative treatment become clear, for these are all dependent upon the persistence and the degree of disturbance of vasomotor equilibrium. Microscopically, my sections show an increase in blood-vessels. The resultant hyperæmia produces the rarefaction seen in the X-ray and the replacement of bone and marrow by fibrous tissue seen in tissue sections.

Classification.—Many authors have compared and classified these conditions of the semilunar and scaphoid with the Legg-Perthe's, Osgood-Schlatter's, Koehler's disease and the other osteochondridities. This I believe is incorrect and misleading. The osteochondridities are affections occurring during the periods of greatest and most active growth, and they are incidental to growth. With the cessation of growth these conditions subside. The involvements of the semilunar and scaphoid bones under consideration are affections occurring in fully formed bones in which the growth has been completed, and they are apparently not involved with the processes of ossification in any way whatever. Comparisons with Kümmel's disease of the spine and the absorption occurring in some fractures of the neck of the femur is, I believe, justified, for these conditions seemingly belong to the same category.

Prognosis.—Preiser cites an instance in which three circular areas of halistersis appeared two days after injury. Ten days later these areas coalesced into one defect. This case eventually recovered completely clinically and röntgenographically. Other authors report instances of complete recovery under conservative measures. Hahn records a case in which osteoarthritic changes were present when seen fifteen years later. Kienböck also speaks of late osteoarthritic changes. DeQuervain, quoted by Guye, observed two cases, one for ten and another for twelve years, which showed osteoarthritic changes with concomitant disability. My first case had symptoms for three years, with a marked increase in disability toward the end of this period. My third and fourth cases had a course of disability and discomfort with an intervening period of freedom from symptoms. On the whole, there seems to be sufficient evidence to justify the belief that many cases recover completely, others have periods of either complete or comparative relief from disability, while another group becomes continuously incapacitated.

Treatment.—Hirsch has removed the scaphoid, on making the diagnosis, and has obtained good results. In the hands of other surgeons and myself, good functional results have been obtained. On the other hand, a great number of cases have had relief from conservative treatment in the hands of

other observers and myself. In view of the general experience with these cases, I would advise conservative treatment in the form of physiotherapy and support for a varying period of time. If, in the face of these measures, the condition still persists, operative removal of the offending bones is justified, for the results are, on the whole, good. One must always keep in mind that persistent conservatism will eventually find the wrist involved with osteoarthritic changes and their accompanying difficulties.

Terminology.—As one would expect, the terminology is varied and confusing because of our lack of knowledge and appreciation of the character of this condition. Most commonly the terms "Kienböck's Disease of the Semilunar" and "Preiser's Disease of the Scaphoid" are in vogue. This is palpably unsatisfactory. Kienböck calls it "Traumatic Malacia," while Axhausen challenges this: "Not Malacia but Necrosis of the Carpal Semilunar." Preiser at first named this condition "Traumatic Rarefying Osteitis" and later changed it to "Traumatic Nutritional Disturbance." Mikulicz speaks of this as an "Osteodystrophia Systica," while Wollenberg prefers "Local Osteitis Fibrosa." Guye speaks of a "Compression Fracture and Traumatic Softening of the Semilunar." Dudan speaks of "Softening of the Semilunar." Henderson uses the term "Chronic Osteitis of the Semilunar," while Rogers captions his articles "Traumatic Osteitis of the Wrist." Fontaine writes under the heading "Traumatic Atrophy." Lériche speaks of "A Localized Osteoporosis." All of these terms, it seems to me, are unsatisfactory, for they suggest either a pathology that is non-existent, or stress a phase that is not the most important. One cannot speak of an osteitis when there is no evidence of inflammation. Nor can one designate it a malacia or a necrosis when these conditions do not exist. Traumatic nutritional disturbance is not sufficiently inclusive, for it does not indicate the actual pathology. "Traumatic Osteoporosis of the Carpal Bones" is to my mind the most inclusive term, for the primary etiological factor is an injury, and the outstanding pathological state is a patchy thinning and rarefaction. I believe that "Traumatic Osteoporosis of the Carpal Bones" is a more fitting title, as it intimates the cause and the approximate structural changes.

SUMMARY

This contribution deals with a little known disease entity for which the term "Traumatic Osteoporosis of the Carpal Bones" seems most applicable. A résumé of the historical facts, anamnesis, clinical, röntgenological, pathogenic, and pathologic pictures are given. Three cases of involvement of the scaphoid, one of the scaphoid and semilunar, and one of the scaphoid, semilunar, and os magnum, one of the semilunar and os magnum and unciform, and one of the os magnum and unciform are presented in detail. The latter three cases are apparently the only ones recorded showing the affection in the os magnum and unciform. An unusual and hitherto undescribed instance of lateral displacement of the semilunar bone is included.

BIBLIOGRAPHY

[1] Axhausen, G.: Nicht Malacie, sondern Nekrose des Os lunatum carpi. Arch. f. klin. Chir., vol. cxxix, p. 26, 1924.

[2] Baum: Über die sogenannte Ostitis des Os lunatum. Zentralbl. f. Chir., vol. xl, p. 1405, 1913.

[3] Baum, E. W.: Über die traumatische Affektion des Os lunatum und naviculare Carpi. Beitr. z. klin. Chir., vol. lxxxvii, p. 568, 1913.

[4] Becker, Georg: Die isolierte Erkrankung des Mondbeines unter besonderer Berücksichtigung und Unfallbegutachtung. Beitr. z. klin. Chir., vol. xciv, p. 172, 1914.

[5] Castruccio, Rodolfo: Der Bruch des Kahnbeins des Karpus. Beitr. z. klin. Chir., vol. liii, p. 66, 1907.

[6] Destot, Étienne: Injuries of the Wrist. Paul B. Hoeber, pp. 85 and 96, 1926.

[7] Ducourthial, D.: Des lesions traumatique du semi-lunaire, vol. lxv, p. 328, 1927.

[8] Dudan, Alfred: Le ramolissement du semi-lunaire du carpe et ses consequences pratiques. Rev. méd. Suisse Rom., vol. xliii, pp. 656, 726, 1923.

[9] Finsterer, H.: Der isolierte Bruch des Mondbeines. Beitr. z. klin. Chir., vol. lxiv, p. 85, 1909.

[10] Frenkel-Tissot, H. C.: Beiträge zur Frage der traumatischen Ernährungstörung des Os lunatum manus. Fort. a. d. Geb. Röntgen, vol. xxi, p. 536, 1914.

[11] Fontaine, R.: A propos de l'atrophie traumatique du semilunaire (maladie de Kienböck). Rev. de Chir., vol. lxiii, p. 769, 1925.

[12] Gaza: Über die sekundären Veränderungen ("traumatische Malazie") nach Frankturen des Os lunatum und Os naviculare carpi. München med. Woch., vol. lxi, p. 2059, 1914.

[13] Goldsmith, Ralph: Kienböck's Disease of the Semilunar Bone. Annals of Surgery, vol. lxxxi, p. 857, 1925.

[14] Guye, Georges: Die Kompressionsbruch und die traumatische Erweichung des Mondbeines. Deutsche Zeitschr. f. Chir., vol. cxxx, p. 118, 1914.

[15] Haenisch: Bemerkung zu der Arbeit von Kienböck und Preiser. Fort. a. d. Geb. d. Röntgen, vol. xvi, p. 239, 1910–1911.

[16] Hahn, O.: Über zentrale Destruktionsherde des Os naviculare nach Trauma. Bruns Beitr. z. klin. Chir., vol. cxxi, p. 704, 1921.

[17] Henderson, Melvin S.: Chronic Osteitis of the Semilunar Bone. (Kienböck's Disease.) J. Bone and Joint Surg. N. S., vol. viii, p. 504, 1926.

[18] Hering, W.: Zur Frage der Mondbeinmalazie. Zentralb. f. Chir., vol. li, p. 505, 1924.

[19] Heyman, Clarence H.: Osteoporosis Relieved by Sympathectomy. J. A. M. A., vol. lxxxii, p. 1333, 1924.

[20] Hirsch, Maximilian: Eine besondere Form des Kahnbeinbruches im Röntgenbilde. Fort. a. d. Geb. d. Röntgen., vol. xvi, p. 222, 1910–1911.

[21] Houzel, G., et Mouchet, A.: Scaphoïde carpien pommelé. Bull. et. Mém. Soc. Nat. d. Chir., vol. lii, p. 518, 1926.

[22] Kautz, F.: Zur isolierten Malazie des Os naviculare carpi. Fort. a. d. Geb. d. Röntgen., vol. xxxi, p. 258, 1923.

[23] Kienböck, Robert: Über traumatische Malazie des Mondbeins und ihre Folgezustände. Fort. a. d. Geb. d. Röntgen., vol. xvi, p. 77, 1910–1911.

[24] Kurlander, Joseph J.: Chronic Osteitis of the Semilunar Bone (Kienböck's Disease). Ohio State Med. Jour., vol. xxiii, p. 303, 1927.

[25] Lériche, R., et Policard, A.: Les Problèmes de la Physiologie Normale et Pathologique de l'Os. Masson et Cie., Paris, 1926, p. 203.

[26] Lériche, R.: A propos du scaphoïde carpien pommelé. Bull. et Mém. Soc. Nat. Chir., vol. lii, p. 622, 1926.

[27] Mikulicz, J. von: Über cystische Degeneration der Knochen. Verhand. d. Ges. deut. Naturforsch. u. Ärzte, vol. lxxvi, p. 107, 1904.

[28] Mouchet, Albert, et Pichat: Semilunaire "pommelé" ou kystique après traumatisme. Bull. et Mém. Soc. Nat. de Chir., vol. lii, p. 200, 1926. Abstracted in Rev. d'Orthop., vol. xiii, p. 362, 1926.

[29] Moutier, Georges: Fracture par ramolissement du semi-lunaire (malacie traumatique de Kienböck). Rev. d'Orthop., vol. xiii, p. 231, 1926.

[30] Peste: 1843. Quoted by Goldsmith.

[31] Pfitzner: Quoted by Gaza.

[32] Preiser, Georg: Zur Frage der typischen traumatischen Ernährungsstörungen der kurzen Hand und Fusswurzel Knochen. Fort. a. d. Geb. d. Röntgen., vol. xvi, p. 360, 1910–1911.

[33] Preiser, Georg: Über eine typische posttraumatische und meist zur Spontanfraktur führende Ostitis navicularis carpi. Zentralbl. f. Chir., vol. xxxvii, p. 929, 1910.

[34] Preiser, Georg: Über eine typische posttraumatische und zur Spontanfraktur führende Ostitis naviculare carpi. Münchener med. Woch., vol. lvii, p. 1200, 1910.

[35] Preiser, Georg: Eine Typische posttraumatische und zur Spontanfraktur führende Ostitis des Naviculare carpi. Fort. a. d. Geb. d. Röntgen., vol. xv, p. 189, 1910.

[36] de Quervain, F.: Spezielle chirurgische Diagnostik. Leipzig, 8 Aufl., p. 688, 1922.

[37] Rogers, Mark H.: Traumatic Osteitis of the Wrist. Surg. Clin. of N. Am., vol. i, p. 697, 1921.

[38] Rogers, Mark H.: Traumatic Osteitis of the Wrist. Bost. Med. and Surg. Journ., vol. clxxxvi, p. 730, 1922.

[39] Saupe, Erich: Beitrag zur traumatischen Ernährungsstörung des Os lunatum. Bruns Beiträge, vol. cxxviii, p. 187, 1923.

[40] Sonntag: Über Malacie des Lunatum. Fort. a. d. Geb. d. Röntgen., vol. xxx, p. 487, 1923.

[41] Speed, Kellogg: A Text-book of Fractures and Dislocations, with Special Reference to their Pathology, Diagnosis, and Treatment. Lea and Febiger, Phila., 1916, p. 527.

[42] Wollenberg, G. A.: Knochencyste im Os naviculare. Berl. klin. Woch., vol. xlviii, p. 616, 1911.

[43] Wollenberg, G. A.: Über die Preiserschen Navikularezysten. Fort. a. d. Geb. d. Röntgen., vol. xviii, p. 392, 1912.

DERANGEMENTS OF THE KNEE-JOINT*

By Melvin S. Henderson, M.D.

of Rochester, Minn.

From the Section on Orthopedic Surgery, Mayo Clinic

The knee-joint is a compound, hinge-like joint. It is the largest joint in the body and depends for its chief strength on an intricate, but remarkably efficient, system of ligaments. The muscles contribute considerably to stability but are secondary in importance to the ligaments.

The large condylar surfaces of the femur rest on the tuberosities of the tibia in which are slight depressions. The semilunar cartilages contribute appreciably to increasing the depth of these depressions for the reception of the condyles of the femur. The internal lateral and the external lateral ligaments are important structures, but are specialized portions of the capsule. The crucial ligaments, anterior and posterior, are distinct structures placed in the form of a letter X in the middle of the joint, and are important factors in stabilizing the joint. The posterior crucial ligament is of more importance in this discussion, because of its possible bearing on osteochondritis desiccans. It arises posterior to the tibial spines and courses upward, forward and inward, to be inserted into the anterior portion of the external surface of the internal condyle, and prevents the femur from slipping forward on the tibia. The movements of the knee-joint are limited to a single plane. Flexion is inhibited normally by the impact of the calf of the leg on the posterior surface of the thigh, and the heel on the buttock. Hyperextension is prevented by the action of practically all the ligaments: the crucial, the internal lateral and the external lateral, and the posterior part of the capsule to a less extent. There is practically no lateral motion in the knee-joint, the slight amount that is permissible being present when the knee is partially flexed and the leg and foot everted. It is in this position of partial flexion and eversion of the leg and foot that injury to the semilunar cartilages occurs. In this position the internal cartilage is placed in its deepest position in the joint. Outside of ordinary sprains of the knee-joint, the ordinary types of derangements encountered are due (1) to injury to the semilunar cartilage, (2) to loose osteocartilaginous bodies, and (3) to slipping patellas.

Semilunar Cartilages.—The internal semilunar cartilage is injured more frequently than the external, the proportion being variously estimated to be from eight to fifty internal to one external. The reason for this vulnerability of the internal cartilages can be explained on anatomic grounds. The internal meniscus is intimately attached to the capsule, whereas the external is loosely attached. The internal, being thus anchored, cannot slip out of danger when nipped by the condyle, as can the external. When the derangement is due to

* Read before the Tristate Medical Society, January 19, 1928.

the semilunar cartilages, there is almost always a history of injury sustained in some game or active vocation. The patient is usually a male in the active period of life. The pain is acute and severe, so severe that it overshadows all else at the time, and the patient is often unable to give an intelligent description of the type of force and the position of the leg, and so forth, at the time of accident. The usual history, however, is that the foot was everted, the knee partially flexed, and the weight transmitted in such a manner that a strain was placed on the internal lateral ligament. The knee was caught or locked in partial flexion and attempts at extension caused excruciating pain. Effusion in the joint followed. Extension was accomplished only after various manœuvres, either by the patient or by his companions forcefully pulling the leg. Disability was complete for the time being, and effusion persisted for a varying period, from a few days to a few weeks. Usually no relief was experienced until the knee was "put in," the patient feeling the knee reduced with a sort of click or snap.

It is not always easy to distinguish between injuries to the internal and external cartilages, but in most cases, if the external cartilage is involved, the pain is felt on the outer side of the knee and a little posteriorly. In a recent review of a series of 232 cases, at the Mayo Clinic, in which the cartilage was removed, 214 were internal and eighteen external. The histories did not show any difference in the types of injuries sustained that would help in the differential diagnosis of internal and external cartilage. Operative procedures are not necessary unless the derangement cannot be reduced, or recurrent attacks occur.

Fractures of the semilunar cartilage are more common than is ordinarily believed. The term "loose semilunar" is used in America quite frequently, but it is somewhat rare that a loose semilunar cartilage is the cause of the trouble. More often it is a fracture or a tear in the substance of the cartilage. The most common type of tear is the loop, or bucket-handle described by Morison (Fig. 1), in which the internal condyle catches the meniscus, ripping it in the middle three-fifths, and crowding over into the intercondylar notch, a loop which is attached anteriorly and posteriorly. As long as this loop lies in the intercondylar notch, extension cannot be complete. This same type of tear is occasionally seen in the external cartilage. The Röntgen-ray is of no value in the diagnosis, for the semilunar cartilage does not cast a shadow.

In the removal of the semilunar cartilage, various approaches to the knee-joint have been advised, but there is no incision that gives really satisfactory exposure. I [2] have found that in removing the internal semilunar cartilage, it is best to let the leg hang over the operating table at a right angle, using an ordinary straight incision about 3 cm. to the inner side of the patellar tendon, extending from well up on the curved internal condyle of the femur, across the joint line, onto the tuberosity of the tibia. At times, difficulty will be found in freeing the posterior portion of the cartilage, and in such cases, when it seems imperative that this posterior portion should be removed, the posterolateral incision which I have described for removal of

loose bodies from the posterior compartment is of great aid. No fear need be entertained about making both these incisions at the same sitting, because neither interferes in any way with the internal lateral ligament. For the removal of the external semilunar cartilage, the same type of incision on the outer side is preferred.

Loose Osteocartilaginous Bodies.—These loose bodies may be formed by osteoarthritis of the knee-joint, osteochondritis desiccans, or osteochondromatosis. As long as the loose bodies are in the byways of the joint, and are not caught between the ends of the bone, they give little or no trouble.

FIG. 1.—Bucket-handle fracture of internal semilunar cartilage showing the loop in the intercondylar notch.

Osteoarthritis is usually seen in the elderly patient and there may be as many as three, four or five bodies in a joint. They arise from the marginal osteophytes in the angle of reflection of the synovia from the bone. They are usually irregular and rough, and are readily seen in the röntgenograms. As they wander about the joint, they receive nourishment and may increase in size and their surfaces become smoothed by their travels.

Osteochondritis desiccans is somewhat rare and not infrequently bilateral. No really satisfactory explanation has ever been given as to why such a condition exists, but it is more than probable that there is an inherent brittleness in the joint cartilage. Almost invariably, the loose body is formed on the internal condyle of the femur, just anterior and proximal to the insertion of the posterior crucial ligament. (Fig. 2.) Koenig, who first described this condition in 1887, argued that it was due to blocking of the end artery which supplies this region. Freiberg and Woolley, and Ridlon have contributed interesting and valuable articles on the subject. Arising so persistently, as these bodies do, in the area contiguous to the posterior crucial ligament, it would seem that the repeated trauma, caused by tugs and pulls sustained by this ligament, might have something to do with devitalizing the area mentioned. While the area from which the loose body is to arise is undergoing dissection, or throwing off, the patient complains of a feeling of insecurity and weakness in the knee. This prodromal stage may go on for several years before the body is cast off. I have had the opportunity of opening a few knees while this formative process was going on. The area involved has a dead-white appearance, and a line of demarcation can be seen to be gradually forming. On pressing with a dull instrument on this spot, there is a feeling of giving way, almost as though a coiled spring were underneath. In several

instances, I have removed the body from this bed by merely cutting along the margins with the sharp point of a knife. Very often there comes away with this piece of the articular cartilage some of the spongy bone beneath, but as the body wanders about the joint it takes up nourishment, and cartilage gradually extends over the spongy bone.

Osteochondromatosis is a still more rare condition, wherein the synovial membrane takes on the properties of forming these loose bodies. (Fig. 3.) This is best explained by a consideration of the embryology of the part. The component parts of the joint, as we know them in the adult, develop in the embryo from the same mesial layer of the blastema. At one stage there is no differentiation, but as growth takes place in the fetus, differentiation is gradual. In the region of the prospective knee-joint, liquefaction occurs to a certain extent, and a space is formed. Cellular differentiation gradually ensues. The function of certain cells is to form bone, of certain cells to form cartilage and of certain others to form synovial membrane. In a few instances, when adult life is reached, there is a sort of reversion to type, and the synovia is prone to form loose bodies. Most of the bodies of this type are formed in the region of reflection of the synovia from the bone, where we would expect there might be a mix-up in the cells. Occasionally, however, the whole synovia is involved. In several such cases I have noted on opening the joint little teat-like pieces of synovia hanging down, and on the ends of these some fibrous tissue, and on others little cartilaginous knobs. As these knobs become larger and heavier, they eventually break loose and wander about the joint, and undoubtedly increase in size. Great numbers of loose bodies may thus be formed; in some rare cases reported, as many as 1000 had been removed from a single joint. They vary in size from a fifth of a centimetre to two or more centimetres.

FIG. 2.—Osteochondritis desiccans. Loose body on the internal condyle.

The symptoms produced by loose bodies are catching or locking similar to that seen in fractures of the semilunar cartilages, but the locking is transient and not so painful and the patient is usually able to palpate the loose bodies himself. The röntgenogram clinches the diagnosis, the bodies, being

calcareous, cast a shadow. The presence of these bodies is, *per se*, reason for their removal, other things being equal. Single bodies can sometimes be removed under local anæsthesia by transfixing the body and holding it with a sharp needle while it is cut down on. When a considerable number of bodies is present, it is better to split the patella longitudinally, and thus secure good access to the anterior compartment. Much has been said in criticism of the split patellar incision, some claiming that unevenness on the under surface following healing may lead to pain, but the incision has been used in a large number of cases in the clinic with no bad results. When the procedure is indicated I see no reason for hesitating to employ it.

FIG. 3.—Osteochondromatosis. Multiple loose bodies.

Slipping Patellas.—Slipping patellas are usually seen in females, and often there is a history of its occurrence in other members of the family. The dislocation is generally outward, although patients often insist that it is inward, mistaking the prominent internal condyle of the femur for the patella. It is probably due to congenital maldevelopment of the patella, and does not always occur in persons with knock-knee. The pain due to the dislocation is severe and the patient is often unable accurately to describe what happened. Sometimes it is not easy to distinguish, from the subjective history, between a slipping patella and the locking due to semilunar cartilages. One simple test which I have found useful is to have the patient sit so that the heel of her leg can be conveniently placed on my knee; then I gently push the patella a little outward and flex her knee. If a slipping patella is causing the trouble, the patient will usually say that the sensation felt is somewhat similar to that experienced when the giving way of the knee occurred and she will become quite apprehensive if I persist in pushing outward on the patella. Röntgenograms are of no help in diagnosing this condition, except that in some cases a small patella will be noticed.

The treatment for a slipping patella is essentially surgical, and the simple procedure of transferring the insertion of the patellar tendon to the inner side at the midline will prevent dislocation.

CONCLUSIONS

1. The knee-joint owes its stability chiefly to the ligamentous structures, the muscles aiding.

2. Derangements due to intrinsic causes are fractures and injuries to the semilunar cartilages, the internal being injured many times oftener than the external.

3. When the lockings persist, the offending meniscus should be removed.

4. Osteocartilaginous loose bodies produce derangements also, and when present, other things being equal, should be removed. They are due to osteoarthritis, osteochondritis desiccans, or osteochondromatosis.

5. Slipping patellas usually occur in the female, and often there is a familial tendency. The patella always slips outward and can be corrected by transferring the insertion of the patellar tendon well to the inner side of the middle line.

BIBLIOGRAPHY

[1] Freiberg, M. D., and Woolley, P. G.: Osteochondritis Desiccans Concerning Its Nature and Relation to Formation of Joint Mice. Am. Jour. Orthop. Surg., 1910, vol. viii, pp. 477–494.

[2] Henderson, M. S.: Posterolateral Incision for the Removal of Loose Bodies from the Posterior Compartment of the Knee-joint. Surg., Gynec. and Obst., 1921, vol. xxxiii, pp. 698–699.

[3] Koenig: Ueber freie Körper in den Gelenken. Deutsch. Ztschr. f. Chir., 1888, vol. xxvii, pp. 90–109.

[4] Morison, Rutherford: Injuries to the Semilunar Cartilages of the Knee-joint. Clin. Jour., 1913, vol. xlii, pp. 1–7.

[5] Ridlon, John: Osteochondritis Desiccans. Jour. Am. Med. Assn., 1913, vol. lxi, pp. 1777–1779.

MELANOTIC TUMORS*

NONMELANOTIC MELANO-EPITHELIOMAS AND THEIR RELATION TO THE MELANO-EPITHELIOMAS

By Alec Horwitz, M.D.

of Rochester, Minn.

Fellow in Surgery, the Mayo Foundation

From a morphologic standpoint there are three main contentions relative to the origin of melanotic tumors:

1. They arise from the epidermis.
2. They are of mesodermal origin.
3. They are specially characterized cells of mesodermal origin, so-called chromatophores.

Ewing's noncommittal view is tersely expressed: "Theoretical considerations favor the origin of all melanomas from the mesoblastic chromatophore, while histology favors their origin from epithelial cells which have taken on pigmentary function."

More recently investigators have attempted to settle the controversy by study of the embryology, but again, they too differ in their conclusions. Acton in his embryologic studies on the tree frog concludes that in lower animals the melanoblasts form a large continuous sheet under the skin surface layer (primitive pigment sheet) which is closely related to the angioblasts of the cutaneous respiratory capillaries.

Acton believes that the melanoblasts (which form pigment) are primarily in the dermis, and that processes from these cells envelop the basal cells of the rete malpighii, and the pigment diffuses between the cells into the intercellular bridges. He finally concludes that there are two types of cells involved in the melanotic tumors: (1) melanoblasts, giving rise to melanosarcoma, and (2) angioblasts (which with the melanoblasts form the benign moles) giving rise to malignant endotheliomas.

Spencer is a strong protagonist of the epidermal origin of the melanotic tumors. His observations on the ink-sac of the sepia show that epithelial cells form melanin pigment. He further observed that in embryo frogs branched pigmented cells develop in the epidermis and pass downward into the subepithelial tissues. This is in direct contradiction to Acton's observations.

Spencer, in his studies on the eye, states that the pigment of the choroid is developed from the retina either by the ingestion of pigment by the cells of the choroid acting as phagocytes, or the cells have grown down into the choroid from the retina. He explains the distribution of melanin-pigmented

* Abridgment of thesis submitted to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery, September, 1926.

cells in the body by the fact that primarily they developed around the neural tube for protection of the central nervous system. As further development occurred, the pigmented cells first spread into the epidermis and then deeper into the mesoblast of the immediate neighborhood and were then carried along the parietal and visceral layers of the mesoblast along with the outgrowing nerves, especially the sympathetic nerves. Pigment cells, therefore, are present in the epidermis, in the rete malpighii and to a less extent in those of the rete spinosum and the corresponding epithelial cells which produce shafts of hair. While in the dermis, the pigmented cells may occur singly, in patches, or in sheets at varying distances from the epidermis, but they all originate as dendritic cells which have penetrated from the epidermis downward into the dermis either as melanophores (cells that are pigmented) or as melanoblasts (colorless cells) that become pigmented in the dermis. The epidermal cells make the pigment while the mesoblastic cells take up the pigment freed by the breaking down of epithelial cells, as phagocytes.

Smith states that there are two types of melanotic tumors: those arising from the skin, hair follicles, retina, and certain regions of the brain are epithelial in origin while those from the iris and choroid and the "mongolian spot" (pigment spot found over the sacrum in the dark races) are mesothelial in origin.

Dawson gives an admirable review of the various theories of origin of the melanomas, and from his studies concludes that melanotic tumors are epithelial in origin. But he agrees with Broders and MacCarty that it is not necessary to accept Unna's view that nevi are epithelial cells snared off in early embryonal life. He believes rather that the difference in morphology should be regarded as an expression of their capacity for differentiation. Therefore, the changes in the epidermis are the different stages of a process "uniform in its initiation but manifold in its development"; hence the reason for their mimicking the picture of carcinoma, sarcoma or endothelioma. In this view he refers to Marchand and Adami who pointed out the relationship between the "differentiation and both the proliferative and functioning capacities of cells." He maintains that a great many observers are misled by the fact that both epidermal and mesodermal cells may show pigment. But the epidermal cell is the creator of that pigment and the mesodermal cell is only a phagocyte carrying pigment.

Dawson takes issue with Acton's idea about his "primitive vascular pigment sheet" in the tree frog being mesoblastic in origin, because it represents a more mature stage of pigment formation. He cites Kornfeld who found melanoblasts (in the larval stage) in the epidermis. To regard the primitive pigment sheet in the subepidermal tissues in the mature position of mesoblastic origin "disregards the phases of their migration which are coincident with the phases of metamorphosis of the larval amphibian."

Relative to the choroidal tumors which have been cited as a good reason for believing them to be of mesoblastic origin because the choroid is mesoblastic embryologically, he repeats Spencer's observations as well as his own;

namely, that the retinal pigment cells migrate to the choroid and provide the choroid with pigment. He concludes that malignant melanomas arise from carcinomatous changes in actual nevus cells derived from the epidermis and embedded in the dermis, or from a genetic process starting in the epidermis and progressively undergoing morphologic changes as they migrate into the dermis. He summarizes his ideas as to the many differences in morphology as follows: "These varied series of epithelial cell transformations, resulting in tumors of such diverse types, allow of only one interpretation, that of their epidermal origin and an evidence of the amazing power of anaplasia and redifferentiation of the epidermal cell." This is in accord with the earlier statement of Broders and MacCarty that malignant melanotic tumors are due to the "migratory hyperplasia of the basal (regenerative or germinative) layer of the skin and invades the subcutaneous tissues and distant organs."

Metastasis of Melanotic Tumors.—One of the arguments in favor of the epithelial origin of the melanotic tumors is that they metastasize by way of the lymph-channels to the nearest lymph-nodes. But those favoring the mesothelial origin cite the extension of ocular melanomas by way of the blood stream. As a matter of fact both methods of metastasis occur, but more often the lymphatics are involved. It is only when the tumor has grown rapidly that the blood stream is invaded. Miescher applied the dopa reaction to a metastatic growth from melano-epithelioma with positive results, showing that also the metastatic tumor cells are ectodermal in origin.

Acton, who believes in the dual origin of melanotic tumors, asserts that the "melanosarcomas" spread by rapidly eroding blood-vessels while the melano-endotheliomas spread by permeation along the lymphatics.

Cairns describes a primary tumor of the eye with metastasis to the scapula after eighteen years. He says that melanotic tumors frequently reproduce in bone.

Metastasis may occur in any organ, including the heart muscle and the spleen. Tumors of the eye often metastasize to the liver.

Melanin.—In order to understand fully why some tumors of the melanotic type are pigmented and some are not, it would be well to review our knowledge of melanin. Melanin is a term given to "a varied and complex group of pigments which give color to hair, skin and eyes in man, and are responsible for the color of animals, birds and insects." [30]

Physical and chemical properties.—Ewing states that melanin consists of carbon, hydrogen and nitrogen in the ratio of 1:5:5 and varying amounts of sulphur. Acton says that from 8 to 13 per cent. of sulphur is present while Spencer gives sulphur a content of from 5 to 12 per cent. Melanin contains no iron. Heated in strong alkali melanin from melanotic tumors yields indol, skatol, and volatile fatty acids and ether-soluble acid giving a dark blue color with ferric chloride. The chief product is melanic acid. Under dry heat melanin gives off pyrrhol.

Origin.—The theories as to the origin of melanin are at wide variance. Some authors claim that melanin originates from the hæmoglobin of the blood.

But melanin does not contain iron unless there has been an extravasation of blood into the tumor. Meirowsky, Rossle and Staffel (according to Acton) believe that the origin of melanin is from nucleolar matter of melanoblasts. The coincidence of increased melanin and nuclear degeneration is taken by them to be a distinct sign of a causal relationship between the two. Lubarsch believes that the high sulphur content of melanin is an indication of the pigment being connected with decomposition of albumin. But Acton refutes the nuclear degeneration theory by the fact that the most virile cells have the most pigment. Spencer believes that the nuclear degeneration is a pressure-effect of the pigment in the cells.

Many substances have been said to be the mother-substance of melanin; namely, tyrosin (Gessard, Dunham), epinephrin (Jäger, Neuberg, Meirowsky, and others) and tryptophane (Spiegler, Eppinger, Fasal, and others).[8]

Until recently the general belief was that melanin pigment was produced by the action of an oxidase (tyrosinase) found in the epidermal cells, on an oxidizable colorless chromogen (melanogen) which is formed by the metabolic activity of the cell from the protein molecule brought to it. The term "melanogen" originated with Thormählen who found that the urine of patients suffering from melanotic carcinoma darkened on standing.[32] Tyrosinase is so called because of its action on tyrosin and other aromatic bodies containing an hydroxyl group to form melanin bodies. Tyrosinase has been found widely distributed in the tissues. Gessard[30] found it in melanotic tumors of the horse. Artificial melanin has been formed by the action of tyrosinase of Lepidopteran larvæ on tyrosin.

Recently Bloch[6] brought out the dopa reaction by treating fresh skin or frozen sections with a 0.1 or 0.2 per cent. watery solution of 3–4 dioxyphenylalanine for twenty-four hours at room temperature, or 37° C. A positive dopa reaction is due to oxidation (with condensation) of the dioxyphenylalanine into a dark (smoky-gray, dark-brown or very black) pigment, the dopa melanin which may be diffuse or in granules. The site of the positive dopa reaction is restricted to the protoplasm (not the nucleus) of the malpighian cells of the basal cell layer (and in stronger reactions also the prickle-cell layer) the infundibulum of the hair follicle (Follikel trichter) and the cells of the outer root sheath and hair matrix. In other words, the dopa reaction is restricted to cells of ectodermal origin.[9] The positive dopa reaction should be distinguished from a pseudo-reaction which is found in the leucocytes and sweat gland cells of the cutis (mesodermal origin) which is due to oxidation of dopa by phenolase or polyphenoloxydase present in these cells. The form of cell which gives a positive dopa reaction may be the normal epidermal cell or, in stronger reactions, a dendritic type of cell with long branching protoplasmic processes which enmeshes the neighboring cells. The intensity of the dopa reaction is proportional to the strength of the dopa-oxydase present in the cells. This varies not alone according to species of animal or race, but also from cell to cell in the same section. Some of the cells may be darkly pigmented, others only slightly, and still others not at all. Thus it is easy to understand that under pathologic conditions the variations may be even more striking; this is well shown in the skin and hair of the albinos and vitiligo spots where the dopa reaction is absolutely negative because dopa-oxydase is lacking in these cells.[7, 19] This is also true in the white hair-spots in animals. The intensity of the reaction may also be changed by physical, chemical and infectious influences. The reaction is more intensive after exposure to sunlight, Röntgen-rays, thorium-x, quartz lamp, and so forth[22]; it is most intensive in the pigmented nevus and is absent in vitiligo spots.

Bloch [5] went further to prove that this dopa-oxydase is a ferment, that it is an oxidizing ferment, and lastly that it is a highly specific ferment. This intracellular enzyme is labile and its thermolability varies with the individual and the type of animal. At from 57 to 80° it is weakened and at 100° its ferment action is disturbed. It is destroyed by drying, imbedding in paraffin, ammonium sulphate, dilute ethyl alcohol, acids, alkalis (except in very dilute solutions), proteolytic ferments, strong oxidizing or reducing agents, poisons (such as hydrocyanic acid), ether, chloroform and benzol. Röntgen-rays and ultraviolet rays have very little effect. Distilled water and physiologic sodium chloride solution have a detrimental effect after a time.

To prove that dopa-oxydase is specific for 3–4 dioxyphenylalanine Bloch attempted to obtain a positive dopa reaction by using other chemical agents allied to dopa. These substances were divided into two groups, those without and those with a pyrocatechol nucleus. Among the many substances without a pyrocatechol nucleus were tyrosin, hydroquinol, homogentisic acid, tryptophane and others. Among those with a pyrocatechol nucleus were pyrocatechol itself and many substances which were assumed to be intermediate to dopa. All these substances gave a negative dopa reaction and warranted the following conclusions about the substance acted on by dopa-oxydase. (Fig. 1.)

1. It must be a pyrocatechol derivative.

2. Both hydroxyl groups in the dopa must be intact, since substitution by a methoxy group gave a negative dopa reaction.

3. The side-chain must have at least a propionic acid group and an amino group.

This showed that 3–4 dioxyphenylalanine (or a closely allied substance) is closely related to, or actually is the substance acted on by dopa-oxydase to form dopa-melanin, an insoluble pigment. The dioxyphenylalanine employed was either a levorotatory or racemic (synthetic) form and little difference was noted in these experiments except perhaps in the very light reactions it was noted that the levorotatory form gave a stronger reaction.

Dr. Horwitz.

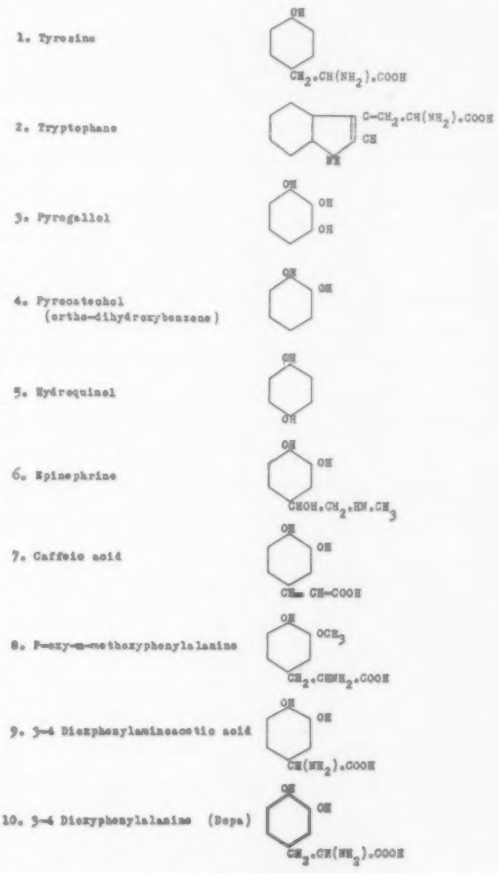


FIG. 1.—Chemical formula.

Fate of melanin.—The fate of melanin in the body has been variously described. Some claim that the pigment enters the blood and is excreted by the glomeruli of the kidney as pigment granules. Others maintain that melanin is changed to colorless melanogen by the liver and then is excreted by the kidney.[26] Nepvin (quoted by Acton), claims that in malignant melanomas mela-

nin is found in the leucocytes in the blood producing melanemia, due to the great number of leucocytes carrying melanin. Acton believes that the achromatic melanogen excreted by the kidney is a reduction substance and not the mother substance of melanin.

Distribution of melanin.—Normally melanin is present mainly in the skin, eye and parts exposed to light.[14] It is also present to some extent in the central nervous system. In the skin it is found in the deeper layers of the rete malpighii and in the branching cells of the corium in the transition zone between the dermis and epidermis, and about the blood-vessels. In the dark races almost all the cells of the basal layer of the rete malpighii are pigmented but in the white races only the areola and mucocutaneous layer of the anogenital region are pigmented.[14] However, that the basal cells of the skin of the white races are potential melanin formers can be easily proved by the bronzing on exposure to the sun.[22] The fetus of both white and colored races have pigment in the basal cells of the rete malpighii, but the cells are depigmented in the white races. In the eye it is present in the cells of the choroid, iris, and ciliary process as phagocytosed pigment, and in the retina where it is a product of a neuro-epithelial cell.[14, 20] The other sites where melanin is present are the brain, cord and occasionally the internal organs. These are due to migration of melanin-producing cells along sympathetic nerves or vessels.

Function of melanin.—The function of melanin pigment is to protect against light, heat and moisture. In the animal it has the function of "adaptation, attraction and offensive and defensive mimicry.[13] Ewing quotes Eppinger who concludes that excess of pigment and its products, especially indol and skatol, are the cause of the overgrowth of cells. Artificial pigment or that extracted from tumors or urine are toxic for animals. Adler believes that the toxic effect may be due to the transformation of melanin into melanic acid which is the toxic agent.

Relation of Nonmelanotic Melano-epithelioma to Melano-epithelioma.—The absence of pigment in the melanotic tumors has been explained by Acton according to his conception of the two-cell theory, as follows: those arising from the melanoblasts, melanosarcoma type, naturally would have pigment while the malignant melano-endothelioma type arising from both angioblasts and melanoblasts would have both pigmented and nonpigmented areas. And since one or the other of the cells may predominate, there would be no pigment if only the angioblasts were present.

Hertzler and Gibson accept Ribbert's chromatophore theory. The melanotic tumors arise from chromatophores whose specific function is to form pigment. These cells, however, often lie dormant, are colorless and give rise to tumors which differ from melanotic tumors only in not containing pigment. They claim that the tumors which tend to ulcerate usually contain little pigment.

Ribbert explains the presence of pigmented and nonpigmented cells side by side in some tumors, as cells which have maturity and immaturity, respectively. The immature cells cannot produce pigment. The nonpigmented part of some tumors may be due, however, to depigmentation.

Thibaudeau and Schreiner in their study of twenty-seven cases of melanotic cancer

believe that there is no relation between the amount of pigment found in the tumor and the degree of malignancy.

Broders and MacCarty, in their review of seventy cases of melano-epithelioma, maintain that the nonmelanotic tumors belong to the same family as the melano-epitheliomas and differ only in the lack of pigment. They have the same cell appearance as the nonpigmented areas of melano-epitheliomas and it is reasonable to believe, these authors state, that if the cells of the nonmelanotic tumors were given the opportunity to differentiate far enough, they would produce melanin.

Broders [3] says that differentiation is a means that malignant tumors have "of putting on the brakes." Melanin production is a form of differentiation. Producing pigment is a function of certain epithelial cells and when the cells proliferate rapidly they follow Marchand and Adami's law,[14] that as a cell carries out its vegetative powers it sacrifices its functioning powers; and we would therefore expect the nonmelanotic melano-epitheliomas to be rapid in growth, and their cells to be more primitive in type.

Another factor that shows that nonmelanotic melano-epitheliomas belong to the same family as the melano-epitheliomas is the presence of melanuria, as reported by various observers in nonpigmented tumors.[23, 25] It must be assumed, therefore, that the chromogen is present but is not acted on by the oxidase either because the cell is too immature to have the oxidase, or if it does have oxidase, not enough opportunity is offered for the oxidase to act on the chromogen, owing to the rapidity of growth.

Pathology.—The tissues in forty-nine cases of nonmelanotic melano-epithelioma were studied microscopically. In certain cases it could be easily demonstrated that malignant changes began in the basal-cell layer of the rete malpighii, either in the papillary or the interpapillary portions of the epidermis. (Fig. 2.) The malignant cells, epithelioid in form, could be seen streaming from one portion of the epithelium into the cutis and then spread out in its upper part. However, in other cases the malignant cells were restricted to the cutis and the epithelial layer was intact. In still others malignant cells from the cutis could be seen invading the subepithelial part of the cutis and even breaking through into the epithelial layer. In only a few cases was lymphocytic infiltration present in the subepithelial layer of the cutis.

The arrangement of the malignant cells was varied. In some it assumed the picture of a fibrosarcoma. (Fig. 3.) The cells were elongated markedly and arranged in whorls. In others an alveolar arrangement was present. (Fig. 4.) In most cases, however, the malignant cells were in solid masses resembling a large or small round-cell sarcoma or massive carcinoma. Only occasionally were the cells arranged in columns or cords. In a few cases the papillary portion of the cutis could be seen invaded by epithelioid cells from the epidermis, and as these cells forced their way into the deeper layers of the cutis they assumed an alveolar arrangement in one area and resembled a fibrosarcoma in an adjacent area, while in still another area the cells appeared definitely carcinomatous. There was little or no intercellular stroma apparent as a rule. Melanin was absent, but blood pigment was present in one case in the form of large irregular patches of a bright sheen. In a few cases the cells were closely applied to young blood-vessels.

The forms of the individual cells were as varied as their arrangement, spin-

dle-formed, round and oval, or markedly elongated. In some they approached more nearly epithelial cells. The nuclei were large in proportion to the rest of the cell, vesicular and hyperchromatic. Some were "one-eyed" (Fig. 5) showing one darkly stained nucleolus but more often there were two or three nucleoli in a meshwork of chromatin. Some of the nuclei appeared stippled, and mitotic figures were more or less numerous. Occasionally vacuoles were

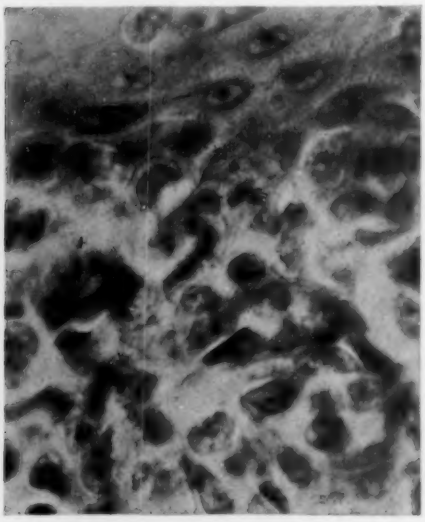


FIG. 2.—Nonmelanotic melano-epithelioma arising in the germinal layer of the skin. The cells are in direct continuity with the stratum germinativum.

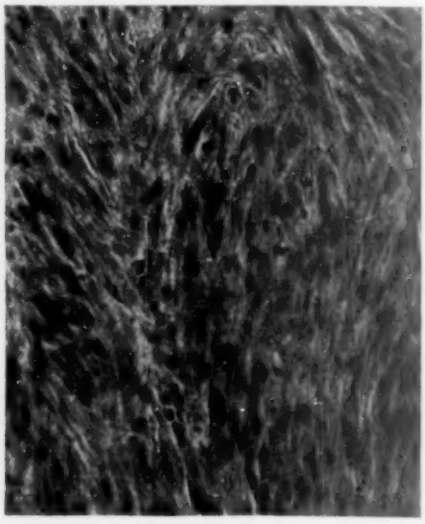


FIG. 3.—Elongated spindle cell simulating fibrosarcoma (X140).

seen in the nuclei. (Fig. 6.) A few sections were found replete with large giant cells with huge nuclei. According to Broders' gradation of epitheliomas all the tumors were graded 4, that is, there was little or no attempt at differentiation. The large cells apparently represent an embryonal state. In general every tumor studied gave the impression of a severe malignancy.

Whatever the form or arrangement of the cells, however, it was apparent that all the tumors were epithelial in origin. The variation in form was only evidence of a different stage of "dedifferentiation" or regression to a more embryonal state of development. This, with the absence of pigment, explains the highly malignant nature of the nonmelanotic melano-epitheliomas.

Clinical Considerations.—Malignant melanotic tumors were first described as occurring in horses in the latter part of the eighteenth century. Laennec first wrote about it in man. The nonmelanotic character of some of the tumors or nonpigmented areas in pigment tumors has been mentioned by many, but reports of cases that were nonmelanotic are few.

It is well known that the primary melanotic tumors may be pigmented and the secondary tumors nonpigmented or only partially pigmented. The reverse is also true.

Etiology.—The primary cause of melanotic tumors is as much a mystery as is the cause of other malignant tumors. Of the predisposing causes many

factors must be considered, namely, trauma, presence of a mole, wart or nevus, site of mole, age, and race.

The history of trauma is in many cases unreliable, but in some cases it is too definite to be ignored, especially when trauma is considered in its broader sense, including in its meaning such factors as chronic irritation, ill-advised and improper surgical procedures or the use of escharotics. In the cases reviewed

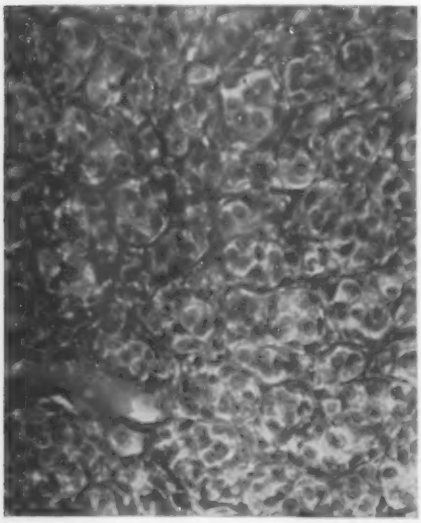


FIG. 4.—Alveolar arrangement of some of the cells (x140).

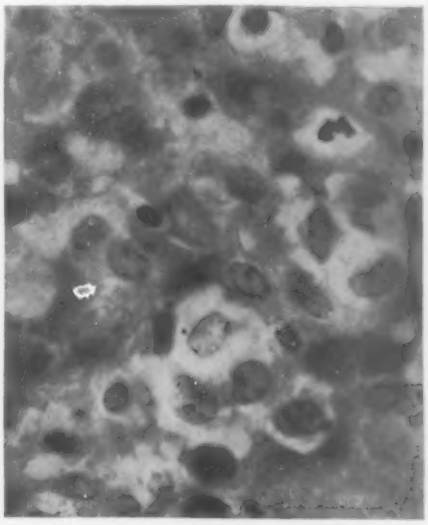


FIG. 5.—"One-eyed" cells and mitotic figures (x500).

here irritation was caused in many instances by improper shoes and in some the activity of the tumor was definitely accelerated after the use of escharotics, ligation, carbon-dioxide snow, and ill-advised operations. Activity seemed to begin in twenty-eight of the forty-nine cases (57.1 per cent.) following injury, irritation, or some means used for the removal of a mole or wart.

Heredity is probably not a factor. Pfingst and Graves report a case of melanosarcoma of the eye in two brothers. They thought it to be coincidental. It is interesting to note that it is considered hereditary in horses. Incidence of carcinoma in the family was found in only six of our cases, but this figure is probably too low. The tumors may occur at any age, but usually past middle life. In this series of forty-nine cases the average age was fifty and three-tenths years, the youngest patient being twenty years and the oldest seventy-seven.

AGE BY DECADES

Years	Patients	Years	Patients
20–29	5	50–59	12
30–39	7	60–69	16
40–49	7	70–79	2
			49

Twenty-seven of the patients were males and twenty-two were females.

The dark races seem to be immune to this type of tumor. It is well known among veterinarians that gray horses after the age of ten become white due to loss of pigmentation in the hair, and then they are prone to melanotic carcinoma. Lubarsch quotes Folger's statistics of 527 carcinomas in 175,745 horses, 226 (42.88 per cent.) were melanotic in type. Of 2274 malignant tumors in 18,113 cadavers of human beings, twenty (0.89 per cent.) were melanotic. Although it is true that the dark races are less prone to malignancy in general, still it is worthy of note that the melanotic tumors are almost unheard of in the dark races in spite of the fact that melanomas are more common in them than in the white races.[32] Seligman noted among the natives of New Guinea that pigmented moles were common on all parts of the body but none had been seen to spread or give to tumor formation. Gilchrist,[32] Sutton and Mallia and Stevenson, Hazen, Carmichael, and Menage describe malignant melanoma in negroes. In my series all patients were white.

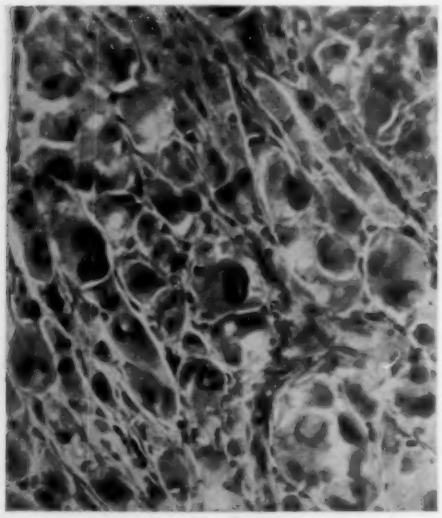


FIG. 6.—Enormous type of cell with large nuclei, some of which are vacuolated (x140).

In eighteen (36.7 per cent.) of the forty-nine cases of nonmelanotic melano-epithelioma the patients gave a history of the presence of a mole, wart, or nevus. In ninety-one cases of melanotic tumors reported by Coley and Hoguet there was a history of mole in thirty-six (39.6 per cent.). In 33 per cent. of Dawson's cases there was a history of mole. In Broders and MacCarty's series of seventy cases, a history of mole, wart or nevus was given thirty-five times (50 per cent.). Apparently the presence of a mole does not indicate whether or not the resulting tumor will be pigmented.

SITE OF THE PRIMARY TUMOR

Site	Cases	Site	Cases
Foot (including ankle)	17	Nose	1
Leg	7	Eye	1
Thigh	2	Ear	1
Groin	2	Cheek	4
Thumb	2	Neck	2
Arm	1	Chest	2
Axilla	3	Back	2
Forehead	1	Vagina	1
			49

It will be noted that of the twenty-eight cases (57.1 per cent.) in which the lesion was on the lower extremities, it was on the foot (the part covered by the shoe) in seventeen (34.7 per cent.). (Fig. 7.) In Coley and Hoguet's series of ninety-one cases the pigmented tumors were on the lower extremities (from groin to toes) in thirty (33 per cent.), and in seventeen (18.7 per cent.) of these, on the foot and ankle; irritation was noted in sixteen of these seventeen cases. In Broders and MacCarty's series of seventy cases of melano-epithelioma, the lesions were on the lower extremities in twenty-nine (41.4 per cent.), and of these, sixteen (22.8 per cent.) were on the foot and ankle. In Dawson's series of thirty-six cases of moles showing transition to malignancy, ten (27.7 per cent.) occurred on the lower extremities and seven (19.4 per cent.) were on the foot. In thirty-one cases in which there was metastasis, Dawson reported nine (29 per cent.) on the lower extremities and of these six (19.4 per cent.) were on the foot. Hertzler and Gibson state that amelanotic types on arm or scalp have not been established. In the series reported here one occurred on the arm of a woman, aged twenty-nine, with metastasis to the axilla. A mole had been present since birth. In one case there was a history of chronic ulcer on the temple.

FIG. 7.—Nonmelanotic melano-epithelioma arising from an irritated spot on the heel, showing metastasis to skin of foot and leg.

Symptoms.—The symptoms in these cases are varied. There is usually a history of a mole or wart which may have been noticed since birth or for many years. This was either bruised, injured or irritated for a long time, especially if occurring on the foot. As was formerly stated, the history of injury is open to question in some of these cases, but on the other hand, in many others the history is too frank to be ignored. A discharging sore or bluish discoloration may follow an injury, or the mole or wart may gradually increase in size and then break down. The discharging sore may heal only to break down again in a short time. It is usually at this time that medical aid is sought and more often than not the true condition is unsuspected by both physician and patient. The area is either excised, or other means of removal such as curettement, ligation, carbon-dioxide snow or escharotics may have been used. This usually is quickly

followed by early recurrence locally and the activity of the tumor is manifested by rapid dissemination to the neighboring lymphatics. It is the multiple recurrences or the involvement of the regional nodes that makes the physician suspect the true significance of the previous symptoms. If the primary trouble occurs in the lymph-nodes the picture is still more confusing, and many times the axillary or inguinal swellings are incised because they are mistaken for abscesses. It is only occasionally that a patient in apparently good physical condition presents himself with generalized metastasis but is worried about the numerous little lumps over the body. Not infrequently there is a latent period between the appearance of the primary lesion and the metastasis. This latent period may be so long as to deceive the patient as to the real relation between the two, and it is only after close questioning that the physician discovers the primary cause, which the patient is only too prone to forget, or considers too trivial to mention.

The latent period may be as long as five or six years; in some instances the history dated back even longer, but this was open to doubt. The following is an example of a case in which there was a latent period of about three years and a rather rapid dissemination in about nine months. A married woman, aged forty-six, came to the Mayo Clinic in 1924 with definite symptoms of mild hyperthyroidism from adenomatous goitre. A thorough general examination did not disclose anything besides the adenomatous goitre and the usual features of hyperthyroidism. Following double resection of the goitre the pathologist's report corroborated the clinical diagnosis of hyperthyroidism. At that time there were no enlarged lymph-nodes, and the pelvic examinations were negative. In February, 1925, nine months later, the patient returned to the clinic complaining of many small lumps over the body of about nine months' duration. She said she had never felt so well in her life; the hyperthyroid symptoms had apparently completely disappeared. On general examination, the skin over the chest and back was peppered with hard firm nodules of various sizes. The right inguinal nodes were markedly enlarged and slightly tender. The pelvic examination disclosed two large immovable masses of firm consistence. After close questioning about the removal of moles and warts, the patient recollected that a small mole had been removed surgically from the anterior surface of the right leg, more than three years before. A healed scar without any sign of local recurrence was found over the anterior surface of the tibia. Pathologic examination of a nodule removed for diagnosis showed nonmelanotic melano-epithelioma. The patient died in November, 1925.

Thirty-five of the forty-nine patients in this series had been operated on or treated by some method for the purpose of removing the growth, previous to their coming to the clinic. The average duration of the lesion was approximately one year and four months. In twenty-two of the cases the lesion was either ulcerating or fungating and in some sinuses at the site of the lesion were discharging. Occasionally the lesion was described as bluish or, at times, brownish. Tenderness, inflammation and pain were not out-

standing features, and were only present, apparently, as a sequel to infection following ill-advised treatment.

In forty-nine cases, twenty-seven (55.1 per cent.) showed regional lymph-node metastasis, while seven (14.3 per cent.) showed generalized metastasis. In four there was only local recurrence.

Diagnosis.—The absolute diagnosis of nonmelanotic melano-epithelioma

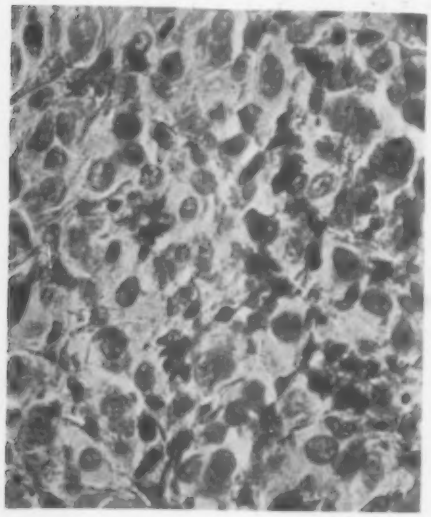


FIG. 8.—Melano-epithelioma showing melanin and alveolar arrangement of cells. Compare to Figure 4 (x200).

FIG. 9.—Squamous cell epithelioma of lower lip graded 4. A matrix and finger-like processes with the columnar cell formation in the advancing portions.

is only possible after microscopic examination. However, in a case of rapidly-growing tumor in which there was a history of a mole, usually on the lower extremities, and especially on the foot, which has been interfered with, or has been subjected to ill-advised treatment, the possibility of a melanotic epithelioma should be considered. If the tumor is very rapid in growth, and possibly if it is ulcerated or fungating in character, is prone to local recurrence or shows early regional lymph-node involvement, it is probably of the nonmelanotic variety.

The microscopic diagnosis of nonmelanotic melano-epithelioma is not simple. The cells may assume so many different forms and the mode of growth may be so varied as to tax the ability of the most competent pathologist. It is not at all unusual for nonmelanotic tumors to be diagnosed as small or large round-cell sarcoma, spindle-cell sarcoma, fibrosarcoma, myxomatous carcinoma or endothelioma. (Figs. 8 and 9.) Still the very fact that the cells are of such diverse shape and form, the mode of growth so varied, the individual cells so completely undifferentiated, and mitosis so frequently encountered, should be sufficient to remind one of the possibility of a nonmelanotic growth. If this diversified picture is found in adjacent areas of the same section, and especially if some definite connection is found with the epidermis, the diagnosis should be quite clear. Occasionally such a tumor may be pigmented due

to the extravasation of blood into the tumor and thus be mistaken for melanotic tumor. But the blood pigment usually has a brighter sheen, is distributed in clumps and irregular patches, and lacks the fine diffuse and regular distribution of melanin pigment.

Prognosis.—The prognosis at the very best is poor. All the tumors of the nonmelanotic variety in our series were graded 4 or 4+, according to Broders' classification. This would mean a good result in only 10 per cent. of the cases. But since in the recent revision of the term, grade 4 malignancy, Broders [4] has stated that it should signify from 75 to 100 per cent. undifferentiated cells and 0 per cent. to 25 per cent. differentiated cells, and since the nonmelanotic tumors are usually almost 100 per cent. undifferentiated, it is only reasonable to expect the results to be very unfavorable.

Forty-four of the forty-nine patients were traced. Thirty-nine (88.6 per cent.) were dead. Most of the patients not traced are no doubt dead because in several generalized metastasis was present when they were last examined. (Tabulation.)

TABULATION

RESULTS OF TREATMENT OF NONMELANOTIC MELANO-EPITHELIOMA

	Cases		Deaths		Patients living	Average post-operative therapeutic life (approximately)†	Range of post-operative therapeutic life (approximately)
	Total	Traced	Number	Per cent.			
Surgery only...	14	11	10	90.9	1 (7 months later)	18.8 months	2 weeks to 6.5 years
Radium or Röntgen-rays only*	5	5	4	80.0	1 (7 months later)	16.8 months	7 to 25 months
Surgery and radium or Röntgen-rays	9	8	5	62.5	3 (2 years 11 months 3 years 8 months 5 years 9 months)	6.1 months	4.5 to 9 months

* Excluding diagnostic biopsy.
† Date of death was not given in three cases in which surgery only was employed and in one in which there was surgery and radium or Röntgen-rays.

Treatment.—With the foregoing prognosis the treatment is far from satisfactory. The use of radium and Röntgen-rays is condemned by some and advocated by others. Wood advises against it in those cases amenable to surgery and Hertzler believes that radium and Röntgen-rays are absolutely contraindicated and worse than useless. Hutchins argues for the removal of moles by electrolysis, caustics or "high potential" treatments because of the good cosmetic result, lack of bleeding and less danger of infection. Owen, and Montgomery and Culver describe a case of melanotic tumor successfully treated by radium.

Thibaudeau and Schreiner report clinical cure in 50 per cent. of melanotic tumors before metastasis appeared and in half of these (25 per cent.) a clinical cure was obtained for two and a half years or more. One case in which the patient was still well after seven years was reported. In another inoperable case in which the tumor was situated at the inner canthus of the eye, the patient was reported to be well after two and a half years following filtered high-voltage Röntgen-ray treatment.

Coley and Hoguet, in their study of ninety-one cases of melanotic tumor, used Coley's serum (mixed toxins of erysipelas and *Bacillus prodigiosus*) with marked retardation of growth of the tumor.

In my series, radium or Röntgen-ray treatment was advised in inoperable cases and many were also advised to use Coley's serum, but lately the serum has not been urged. Operation, in the form of wide excision by cautery, amputation (if on a limb) with removal of the regional lymph-nodes, or irradiation of the regional lymphatics, was resorted to in cases amenable to treatment.

Desjardins [12] believes that the melanotic tumors are the most resistant variety of neoplasms but that occasionally certain cases do respond to radiotherapy. But in order to obtain any such response he maintains that treatment must be thorough. In some cases he has obtained encouraging and sometimes quite satisfactory results.

If a more favorable outlook is to be established stress must be placed on treatment which is anticipatory rather than remedial. "Cosmeticians" and beauty parlor "experts" have learned to give moles a wide berth. The mole has acquired the pseudonym of "touch-me-not" spot, from those who have had bitter experience with it. However, a mole which is constantly irritated, especially one on the lower extremities, and more especially one on the foot, should be removed by a wide excision, preferably by cautery, including the deep fascia and subcutaneous tissue. Any mole which is degenerating or showing evidence of beginning activity, such as bluish discoloration, or gradual increase in size, should be accorded the same treatment. And if unfortunately the mole shows beginning malignancy, then excision alone is not sufficient. The only safe treatment is complete removal of the regional lymph-nodes, as would be done in the case of a small nodule of carcinoma in the breast. New and Hansel do not believe that "removal of the glands of the neck in an attempt to block the growth from metastasizing is of value; nor is a block dissection of value when a gland of the neck is involved."

CONCLUSIONS

1. The nonmelanotic tumors belong to the same family as the melano-epitheliomas and differ only in the absence of pigment.

2. The absence of pigment in these tumors is evidence of their rapid growth, for melanin production is a form of differentiation.

3. Histologically it can be demonstrated that malignant changes begin in the basal-cell layer of the rete malpighii.

4. The arrangement and form of the individual cells are quite varied, and are evidence of a different stage of "dedifferentiation."

5. According to Broders' classification all of the forty-nine cases of non-melanotic melano-epithelioma occurring in the Mayo Clinic are of the grade 4 type.

6. A clinical review of these cases shows that they usually occur when the patient is past middle life. More than half of the growths are on the lower

extremities, especially on the foot, and in more than a third there is a history of a mole, nevus or wart.

7. The prognosis, as would be expected from any malignant growth graded 4, is very poor.

8. Prophylactic excision of moles, especially those occurring on the lower extremities, and wide excision by cautery, with eradication of regional lymph-nodes in moles showing malignant change, is advocated. Radium and Röntgen-rays should be used as an adjunct to surgery and in those cases not amenable to surgery.

BIBLIOGRAPHY

[1] Acton, H. W.: Melanotic Growths; Researches on the Alterations in the Melano-Blastic Layer of Vertebrates, to Explain the Structure and Origin of These Growths. Indian. Jour. Med. Res., 1921–1922, vol. ix, pp. 464–495.

[2] Adler, Oskar: Beiträge zur Melanomfrage. Über melanotische Pigmente. Wien. Arch. f. inn. Med. 1924, vol. vii, pp. 47–50.

[3] Broders, A. C.: The Grading of Carcinoma. Minn. Med., 1925, vol. viii, pp. 726–730.

[4] Broders, A. C.: Cancer's Self Control. Med. Jour. and Rec., 1925, vol. cxxi, pp. 133–135.

[5] Broders, A. C., and MacCarty, W. C.: Melano-epithelioma. Surg., Gynec. and Obst., 1916, vol. xxiii, pp. 28–32.

[6] Bloch, B.: Das Problem der Pigmentbildung in der Haut. Arch. f. Dermat. u. Syph., 1917, vol. cxxiv, pp. 129–208.

[7] Bloch, B.: Zur Pathogenese der Vitiligo. Arch. f. Dermat. u. Syph., 1917, vol. cxxiv, pp. 209–232.

[8] Bloch, B.: Chemische Untersuchungen über das spezifesche pigmentbildende Ferment der Haut, die Dopaoxydase. Ztschr. f. physiol. Chem., 1916–1917, vol. xcviii, pp. 226–254.

[9] Bloch, B.: Über die Entwicklung des Haut und Haarpigmentes beim menschlichen Embryo und über des Erloschen der Pigmentbildung im ergrauenden Haar (Ursache der Canities). Arch. f. Dermat. u. Syph., 1921, vol. cxxxv, pp. 77–108.

[10] Cairns, F. D.: Metastatic Melanoma of Scapula. Brit. Jour. Surg., 1922–1923, vol. x, pp. 290–293.

[11] Coley, W. B., and Hoguet, J. P.: Melanotic Cancer with a Report of Ninety-one Cases. Annals of Surgery, 1916, vol. lxiv, pp. 206–241.

[12] Desjardins, A. U.: Personal Communication.

[13] Desjardins, A. U.: Common Misconceptions in Radiotherapy. Surg., Gynec. and Obst., 1926, vol. xlii, pp. 64–69.

[14] Dawson, J. W.: The Melanomata, Their Morphology and Histogenesis. Edinburgh Med. Jour., 1925, vol. xxxii, pp. 501–732.

[15] Ewing, James: Neoplastic Diseases. Philadelphia and London, Saunders, 1919, pp. 849–868.

[16] Hertzler, A. E.: Melanoblastoma of the Nail-bed (Melanotic Whitlow). Arch. Dermat. and Syph., 1922, vol. vi, pp. 701–708.

[17] Hertzler, A. E., and Gibson, E. T.: Melanoblastomas of the Foot (Chromatophoroma, Melanoma, Melanosarcoma). Annals of Surgery, 1914, vol. lx, pp. 88–102.

[18] Hutchins, M. B.: Should All Pre-cancerous or Cancerous Lesions of the Skin be Excised? Jour. Cutan. Dis., 1916, vol. xxxiv, pp. 27–29.

[19] Kissmeyer, Arne: Studies on Pigment with the Dopa-reaction, Especially in Cases of Vitiligo. Brit. Jour. Dermat. and Syph., 1920, vol. xxxii, pp. 156–162.

[20] Knight, Mary S.: A Critical Survey of Neoplasms of the Choroid. Am. Jour. Ophth., 1925, vol. viii, pp. 791–801.

[21] Lubarsch, O.: Zur vergleichenden Pathologie der melanotischen Gewächse. Med. Klin., 1920, vol. xvi, pp. 195–199.

[22] Lutz, W.: Zur Kenntnis, der biologischen Wirkung der Strahlen auf die Haut, mit spezieller Berucksichtigung der Pigmentbildung. Arch. f. Dermat. u. Syph., 1917, vol. cxxiv, pp. 233–296.

[23] Malejan, H. M., and Russell, V.: Report of a Case of Melanotic Sarcoma. Jour. Mich. State Med. Soc., 1917, vol. xvi, pp. 321–323.

[24] Miescher, G.: Ein Beitrag zur epithelialen Genese der malignen Melanome der Haut. Centralbl. f. allg. Path. u. path. Anat., 1919–1920, vol. xxx, pp. 353–364.

[25] Monteith, S. R.: Report of a Case of Melanoma. New York Med. Jour., 1920, vol. cxii, pp. 252–254.

[26] Montgomery, D. W., and Culver, G. D.: A Melanotic Tumor of the Lip Treated with Radium. Arch. Dermat. and Syph., 1920, vol. ii, pp. 5–6.

[27] New, G. B., and Hansel, F. K.: Melano-epithelioma of the Palate. Jour. Am. Med. Assn., 1921, vol. lxxvii, pp. 19–22.

[28] Owen, A. K.: A Case of Melanosarcoma Treated with Röntgen-rays. Am. Jour. Roentgenol., 1924, vol. xi, pp. 335–336.

[29] Pfingst, A. O., and Graves, Stuart: Melanosarcoma of the Choroid Occurring in Brothers. Arch. Ophth., 1921, vol. i, pp. 431–439.

[30] Smith, D. T.: The Pigmented Epithelium of the Embryo Chick's Eye Studied in Vivo and Vitro with Special Reference to the Origin and Development of Melanin Pigment. Bull. Johns Hopkins Hosp., 1920, vol. xxxi, pp. 239–246.

[31] Smith, D. T.: Evidence Showing the Existence of Two Distinct Types of Pigment Cells Capable of Giving Rise to Melanotic Tumors. Bull. Johns Hopkins Hosp., 1925, vol. xxxvi, pp. 185–198.

[32] Spencer, W. G.: Bradshaw Lecture on Melanosis (Melanin: Melanoma: Melanotic Cancer). Brit. Med. Jour., 1923, vol. ii, pp. 907–913.

[33] Sutton, L. A., and Mallia, W. M.: Malignant Melanoma in the Negro. Arch. Dermat. and Syph., 1923, vol. viii, pp. 325–343.

[34] Thibaudeau, A. A., and Schreiner, B. F.: Histological Study of Melanotic Tumors and the Effect of Radiation on Them. Jour. Cancer Res., 1025, vol. ix, pp. 503–504.

[35] Wood, F. C.: Nelson Loose-Leaf Medicine. New York, Nelson and Sons, 1920, vol. iii, p. 181.

MALIGNANT LYMPHOCYTOMA

By Philip G. Silver, M.D.

of Montreal, Canada

From the Surgical Service of Dr. E. M. Eberts, at the Montreal General Hospital

The purpose of this paper is to report two unusual cases of malignant lymphocytoma, and at the same time to plead for greater uniformity in the nomenclature of lymphoid tumors.

The first case is one of malignant lymphocytoma primary in the small intestine, a condition of sufficiently rare occurrence to warrant its being placed on record. Only one other such case has occurred in the Montreal General Hospital during the past fifteen years.

The second case is one of malignant lymphocytoma of the cervical lymph glands, with an extradural spinal metastasis occurring nine years after the first appearance of a tumor in the neck. The extreme rarity of spinal metastases of lymphoid tumors, and the long duration of this disease in this case, are two features of unusual interest.

Tumors of lymphoid tissue include simple inflammatory hyperplasias, rapidly growing neoplasms, and a variety of intermediate types.

Inflammatory hyperplasias are of frequent occurrence, and are the physiological response to an irritant, bacterial or toxic, brought to the tissue by afferent lymph or blood-vessels.

Neoplastic tumors are the result of an overgrowth of atypical lymphoid cells. They are of relatively uncommon occurrence and their etiology is obscure.

Also of unknown etiology are such conditions as lymph-adenoma, leukæmia, and pseudoleukæmia, which produce a more or less diffuse enlargement of lymphoid tissues and may for this reason lead to difficulties in differential diagnosis.

It is unfortunate that there is such a lack of uniformity in the nomenclature of lymphoid tumors, but this may be partly explained by the lack of knowledge as to their etiology. Since an etiological classification is rendered impossible, the most satisfactory one is based on histogenesis and structure.

The following is the classification of Ewing[1]: There are three elements in lymphoid tissue capable of giving rise to lymphoid tumors, namely: (1) lymphocytes, (2) reticulum cells of follicles and pulp, (3) endothelial cells of pulp and cavernous sinuses. According to histogenesis, tumors of lymphoid tissues are classified as follows:

MALIGNANT LYMPHOCYTOMA

Origin	*Anatomical type*	*Clinical type*
(1) Lymphocytes	Lymphocytoma	Simple lymphoma Tuberculous lymphoma Lymphatic leukæmia Pseudoleukæmia Malignant lymphocytoma (lymphosarcoma)
(2) Reticulum cells	Large round-cell hyperplasia or neoplasia	Granuloma malignum Myeloid leukæmia Hodgkin's sarcoma Large-cell sarcoma (lymphosarcoma)
(3) Endothelial cells	Endothelial hyperplasia or neoplasia	Endothelial hyperplasia of tuberculosis, etc. Endothelioma

The description of two cases of slowly growing lymphosarcoma constitutes the basis of this paper, and further discussion will be limited to this type of tumor.

Lymphosarcoma arises in situations where lymphoid tissue abounds. It is a malignant neoplasm which is locally destructive and which tends to extend by permeation of lymphatics. It arises from an overgrowth of atypical lymphoid cells and may be of two types, a malignant lymphocytoma arising from lymphocytes, or a large-cell sarcoma arising from reticulum cells.

Structurally, lymphosarcomata are characterized by a diffuse overgrowth of lymphoid cells enmeshed in a fine reticulum. The structure varies somewhat according to the rapidity of the growth of the tumor. In rapidly growing tumors there is very little reticulum, the nuclei of the tumor cells are compact and hyperchromatic, and many mitotic figures are seen. In more slowly growing types, the reticulum may be thickened in some areas, even with a tendency to fibrosis, while the nuclei are more vesicular, less pyknotic, and show fewer mitotic figures.

CASE I.—Hospital record M. G. H. No. 2070–1927. H. G., male, age fifty years, was admitted into the Medical Service at the Montreal General Hospital on April 13, 1927.

Complaints.—1. Pain in the epigastrium. 2. Vomiting. 3. Loss of weight and strength.

Personal History.—He had been born in England but had lived in Canada since 1904. He was a stationary fireman; was unmarried; smoked heavily; drank moderately; and had had no serious illnesses apart from a Pott's fracture of the left ankle, and a fracture of the olecranon process of the right ulna.

Family History.—Irrelevant.

Present Illness.—In January, 1927, diarrhœa developed and became rapidly worse until the patient was passing twenty fluid stools daily. He stated that he had never noticed any blood in the stools but that on one occasion he observed a piece of tissue three inches in diameter which contained small "veins." There was nausea and vomiting, occurring daily about one-half hour after the midday meal. He stopped work and was given a remedy which checked the diarrhœa and vomiting.

Shortly before admission, severe cramp-like pain developed in the epigastrium after eating, lasting for a few minutes. More recently the pain had become more frequent,

occurring at irregular intervals and bearing no relation to the taking of food. The pain was always situated in the epigastrium and, for five days before operation, was so severe as to require the administration of morphia each night.

The appetite had remained good, but the patient was afraid to eat for fear of provoking pain. He lost fifty pounds in weight during the three months prior to admission. There was a corresponding loss of strength.

Condition on Admission.—The patient was emaciated. The temperature was normal; the pulse was regular, rate 72; and the respiratory and cardiovascular systems were negative. There was no generalized glandular enlargement. The abdomen was scaphoid. The recti muscles were tense in the upper half of the abdomen. Tenderness was present immediately above the umbilicus. No organs or masses were palpable. The abdomen was tympanitic throughout.

FIG. 1.—Barium series, six-hour plate, showing distention of small bowel and a step-ladder effect in the arrangement of the coils of the ileum.

Laboratory Tests.—Urine—negative. Stools—no visible or occult blood. Blood examination—3,500,000 erythrocytes, 7150 leucocytes, 80 per cent. hæmoglobin.

X-ray examination of the gastro-intestinal tract following the administration of a barium meal, showed in the six-hour plate (Fig. 1) a marked distention of the small bowel, and a "step-ladder" arrangement of the coils of the ileum due to a partial obstruction.

The diagnosis of a partial obstruction of the small bowel was made, and the patient was transferred to the Surgical Service of Dr. E. M. Eberts.

April 26, 1927, the abdomen was opened under ether anæsthesia. From a point six inches above the ileocæcal valve, and extending proximally for a distance of two feet, the ileum was found to be of a purplish color. Its walls were greatly thickened, rigid, and in places covered with a greenish-yellow, translucent, gelatinous exudate. There were numerous enlarged lymph glands in the mesentery. The involved portion of the ileum, together with its mesentery, was resected, and an end to end anastomosis performed.

The patient had an uneventful recovery. He received one X-ray treatment before leaving hospital on May 27, 1927. Six months later he was enjoying perfect health, had gained fifty-three pounds in weight, and there was no evidence of recurrence.

Pathology.—(M. G. H. S–27–423.) The specimen consists of a portion of ileum seventy centimetres in length, together with its mesentery. (Fig. 2.) The walls are thickened and œdematous. The mucosa presents a pebbled appearance and there are multiple minute areas of superficial ulceration. Along the line of mesenteric attachment

of the intestine there are numerous yellowish, firm, oval lymph-nodes, varying from one-half to one centimetre in diameter. Transverse section of the bowel shows macroscopically, marked infiltration of the muscularis with grayish-yellow tissue. In the root of the mesentery there are a number of large, firm, discrete lymph-nodes, which on section bulge from their capsules.

Histological Structure.—The intestinal mucous membrane is normal except for areas of superficial ulceration which occur between low papillæ projecting into the intestinal lumen. (Fig. 3.) These projections are the result of local aggregations of tumor cells in the submucosa, and they give to the mucosa its pebbled appearance.

The submucosa is largely replaced by tumor tissue

FIG. 2.—Drawing of a portion of the ileum with its mesentery. The intestine has been laid open. Its walls are thickened, and the mucosa has a pebbled appearance due to the presence of multiple small tumors of the submucosa. The mesentery contains many firm, discrete, enlarged lymph-nodes.

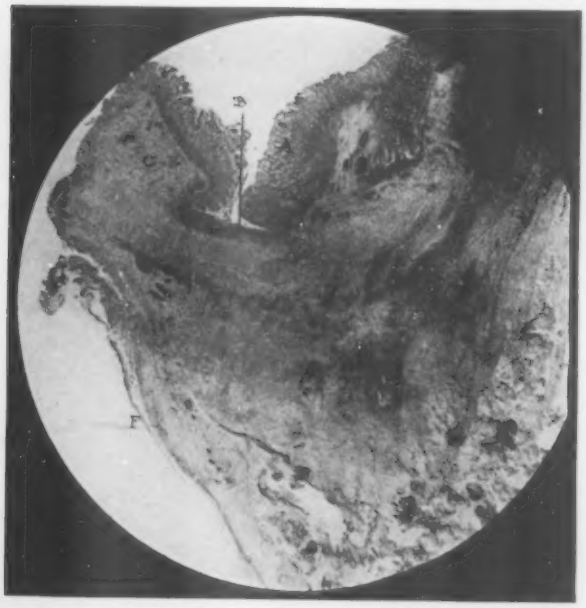


FIG. 3.—Low power microphotograph of section through the intestinal wall at mesenteric attachment showing: A, normal mucous membrane; B, superficial ulceration of mucosa; C, thickened submucosa; D, muscular coats of intestine; E, nests and cords of tumor cells extending from submucosa through the muscular coats and invading the fatty tissue of the mesentery; F, peritoneum.

which is not of a uniform structure. In some areas there are nests and cords of tumor cells with little or no stroma, while in other areas there is a dense fibrous tissue in which there are few tumor cells. From the submucosa, columns of tumor cells are seen invading the muscular coats, producing a wide separation between the muscle bundles. (Fig. 4.)

The tumor invasion extends through the muscular coats into the fatty tissue of the mesentery, which is thickened by local and diffuse tumor cell aggregations, and by an increase of fibrous connective tissue. The structure of the mesenteric lymph-nodes is completely obliterated by invasion with tumor cells. The peritoneum is normal.

The tumor cells are

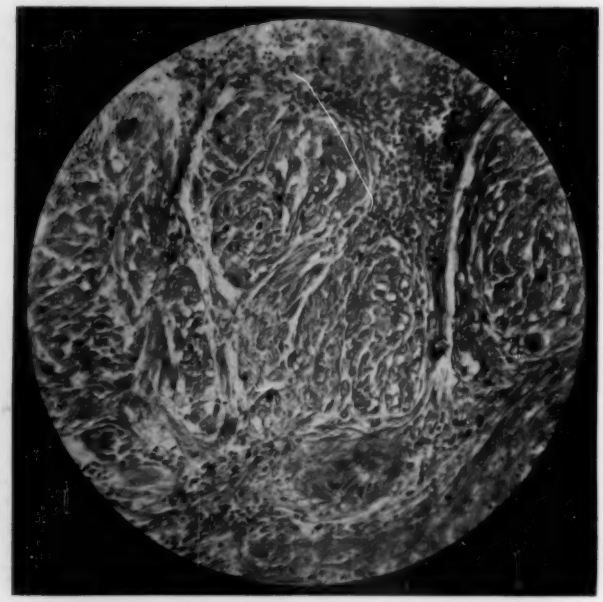


FIG. 4.—Section through circular muscle coat of ileum showing separation of muscle bundles by columns of invading tumor cells.

small, but vary somewhat in size. They are irregularly round, with a small amount of clear cytoplasm and centrally placed nuclei. The nuclei are round but show considerable variation in structure and staining reaction. The majority do not stain deeply, but in these, central strands and granules, and a definite peripheral chromaffin ring are to be seen. Other nuclei are homogeneous, deeply stained, and without visible structure.

In the more cellular areas (Fig. 5) there is a fine fibrillar stroma. In other areas the stroma consists of a fairly mature though newly formed fibrous tissue which contains relatively few tumor cells.

Diagnosis.—Malignant lymphocytoma (lymphosarcoma).

Discussion.—Lymphosarcoma of the intestine is most frequent during the third and fourth decades of life. It is usually characterized by an insidious onset, but some cases have been reported of acute onset simulating appendicitis. Ochsner[3] describes the early symptoms as follows: "Indefinite abdominal pain which is persistent and not relieved by rest or starvation. The pain is not well localized, is colicky, and often associated with anemia and cachexia." Symptoms of partial obstruction are not uncommon, and acute obstruction occasionally occurs, due to an intussusception at the site of the tumor. A palpable mass is sometimes present within the abdomen, and, when present, greatly facilitates the diagnosis.

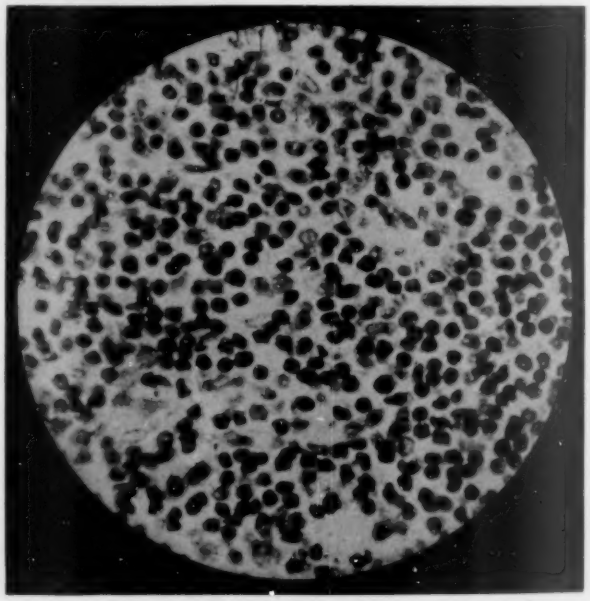


FIG. 5.—High power microphotograph of section from mesenteric lymph-node showing small round tumor cells and fibroblasts.

Lymphosarcoma of the intestines most commonly affects the lower portion of the ileum. Beginning as a localized thickening of the submucosa, areas of ulceration of the mucosa not infrequently result. The tumor may extend through and destroy the muscular coats, and appear beneath the serosa. Metastases to the regional lymph-nodes occur early.

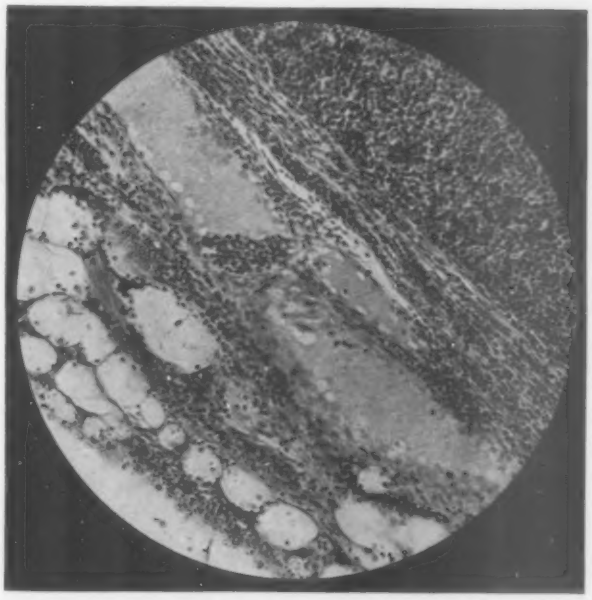


FIG. 6.—Section of tumor of neck removed at first operation (1921) showing the periphery of a lymph-node whose structure is obliterated. The capsule and surrounding tissues are invaded by small round cells of the lymphoid series.

Aneurismal dilatation of the intestine is frequent, and Graves [2] has offered the explanation that this may be due either to an early destruction of the muscle fibres with subsequent dilatation resulting from an accumulation of fæces, or to the early involvement of the submucosa with effect on its plexus of nerves.

Stenosis of the bowel is less common. When it does occur, it is usually the result of fibrous tissue contraction which takes place late in the disease. With the progress of the disease, a large segment of the bowel may become involved, and partial obstruction occur as a result of the loss of peristalsis. A rare cause of stenosis is the presence of a polypoidal tumor mass protruding into the lumen of the intestine. Such a case was reported by De Noyelles.[4]

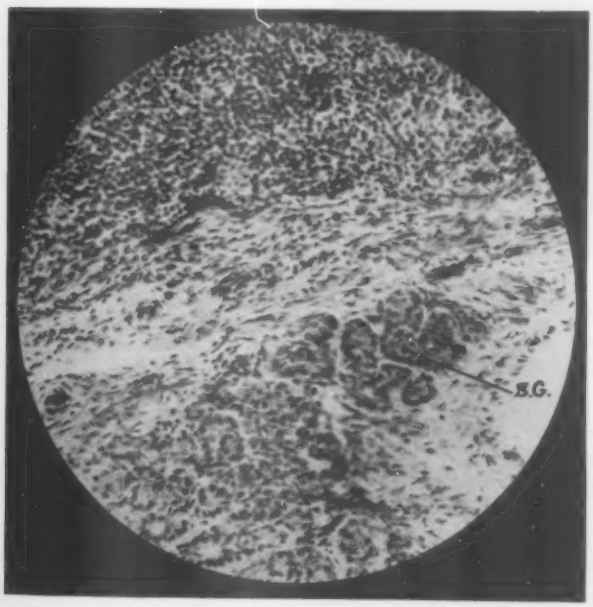


FIG. 7.—Section of tumor of submaxillary region removed at second operation (1924). Submaxillary salivary gland (S.G.) is invaded and partially replaced by tumor composed of small round cells.

Treatment.—The method of treatment which gives promise of yielding the best results is the thorough excision

of the tumor at the earliest possible date, following by irradiation. The value of radium in the treatment of lymphosarcoma is unquestioned. Bloodgood [5] states: "The one lesion of which, as far as I know, surgery has never accomplished a cure, is lymphosarcoma of the lymph glands, and apparently radium has done so." In an article on lymphoblastoma, Minot [6] says that "irradiation is of great value. It alleviates the symptoms, decreases the size of the lesions, and improves the patient's efficiency in spite of the fact that it does not appear to influence importantly the duration of the disease."

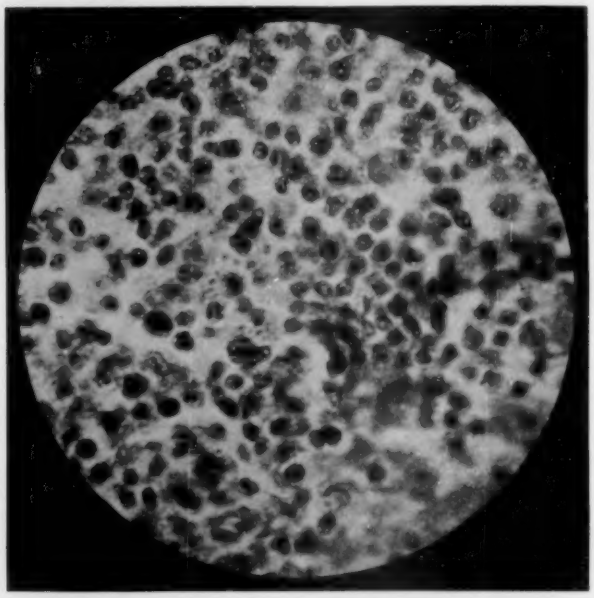


FIG. 8.—High power microphotograph of section from tumor of submaxillary region removed at second operation (1924), showing tumor cells and a small number of reticular cells.

Prognosis.—In untreated intestinal cases, the condition becomes rapidly fatal. Treatment offers no prospect of a permanent cure, but may prolong the patient's life for several months. Rare cases are reported of patients who have lived three years after operation.

CASE II.—Hospital record M. G. H., No. 4275–1927. G. H., male, age thirty-nine years, was admitted into the Surgical Service of Dr. E. M. Eberts at the Montreal General Hospital on August 9, 1927, complaining of weakness in the legs, and pain encircling the chest.

Personal History.—He had always lived in Canada, and with the exception of frequent attacks of tonsillitis, his general health had been good. Married at the age of twenty-five, his wife had had no children and no miscarriages. There was no history of lues. He was employed as a railway shop foreman. He was a moderate smoker, and but seldom took alcoholic stimulants.

Family History.—Irrelevant.

Present Illness.—In 1918, a tumor appeared in front of the left ear, and one year later a similar swelling developed behind the angle of the jaw on the left side. Soon after this, masses were noticed behind the sternomastoid muscle on the left side of the neck. In 1920, surgical advice was sought, and examination showed the tumors to consist of enlarged lymph glands which were freely movable, discrete, moderately firm, and not tender. Their removal was advised, but not until 1921, three years after the onset of the disease, did the patient consent to operation. At operation, the glands were found to be slightly adherent to the neighboring tissues, firm, and of a yellowish-gray color on cut section. Following adenectomy, he received five X-ray treatments.

Pathology.—(M. G. H. S–21–578.) Microscopic examination shows a diffuse overgrowth of small round cells with pyknotic nuclei, producing complete obliteration of

the normal structure of the lymph glands. There is definite invasion of the capsule and of surrounding tissues. (Fig. 6.) Diagnosis: Lymphosarcoma.

In June, 1924, three years after the first operation, or six years after the onset of the disease, a mass appeared on the left side of the neck anterior to the sternomastoid muscle. Under local anæsthesia this was removed, and found to consist of several enlarged lymph glands similar in gross appearance to those previously removed.

Pathology.—(M. G. H. S-24-602.) Microscopic examination (Fig. 7) reveals a compact tumor mass made up of small round cells with scanty stroma, invading the fixed tissues. In the tissue removed is a portion of submaxillary salivary gland which is diffusely invaded and partially replaced.

The characteristic tumor cell (Fig. 8) is small and round, with little cytoplasm. The nucleus is pale, but shows a sharply staining granular network and peripheral chromaffin ring. Intermingled with these cells are a small number of reticular cells. Diagnosis: Lymphosarcoma.

FIG. 9.—X-ray of spine following injection of lipiodol into the cysterna magna, showing obstruction of the spinal canal at the level of the second dorsal vertebra.

He was again given a course of five X-ray treatments, and enjoyed comparatively good health until December, 1926, two and a half years after the second operation, when a feeling of tightness and pain in the left side of the chest was complained of. Physical examination was negative. In June, 1927, the pain became localized to the left axilla and scapular region. Stereoscopic X-ray examination of the thorax eliminated the probability of a mediastinal tumor. The pain gradually increased in severity, became bilateral, and, at the beginning of August, 1927, was described as being situated in the thoracic vertebræ and radiating from there around the chest under the arms. During the week prior to admission (August 9, 1927), there had been rapid loss of power in the legs.

Condition on Admission.—The patient was well developed but had apparently lost considerable weight recently. The skin and mucous membranes showed a moderate degree of pallor. The temperature was normal; the pulse was regular, rate 84; and the respiratory, cardiovascular, and digestive systems were negative.

Nervous System.—Report of examination by Dr. F. H. Mackay, neurologist to the Montreal General Hospital, is as follows: "The cranial nerves are normal. The upper extremities are normal in movement and coördination, but the lower extremities show a marked muscular weakness and only slight movement of the feet and toes can be obtained. The sense of position and vibration is lost in the lower extremities. There

is marked hypoæsthesia and hypoalgesia over the trunk and lower limbs from the level of the fourth thoracic segment.

Reflexes	Right side	Left side
Knee jerks	+++	+++
Ankle jerks	++	++
Plantar	extension	extension
Abdominal	0	0
Clonus	+	+

"*Lumbar Puncture.*—Spinal pressure of 110 mm. of water, showing no alteration on jugular compression. The spinal fluid was of a slightly yellowish color, contained 345 cells per cubic mm., and gave a +++ globulin reaction. Diagnosis: A transverse lesion of the spinal cord from pressure."

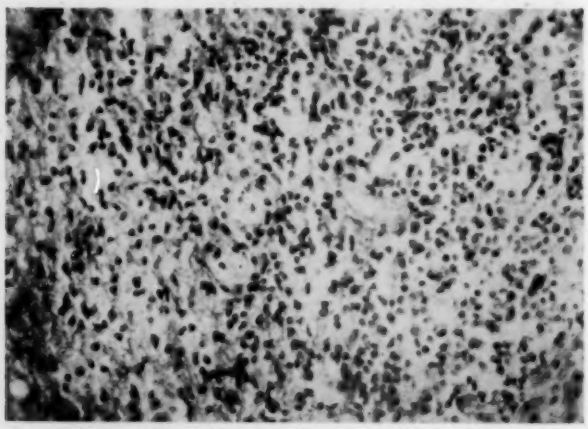


FIG. 10.—Low power microphotograph of section from extradural tumor of spinal canal removed in 1927, showing tumor to be composed of small round cells with areas in which reticulum and capillaries are abundant.

X-ray.—(Fig. 9.) Following the injection of 1 c.c. of lipiodol into the cisterna magna, X-ray showed an obstruction at the level of the second dorsal vertebra (fourth dorsal segment).

Spinal fluid Wassermann and colloidal gold reactions were negative.

Diagnosis.—A transverse lesion of the spinal cord from pressure of a tumor situated at the level of the second dorsal vertebra.

Operation.—August 25, 1927, under intratracheal ether anæsthesia, the spinous processes and laminæ of the first four thoracic vertebræ were removed. A flat tumor 3 cms. in vertical diameter, and 1 cm. in transverse diameter, was found lying at the level of the second dorsal vertebra. A moderate degree of compression of the cord was present. The tumor was not encapsulated, was extremely friable, and did not apparently infiltrate the dura or the bone. It was readily removed, and the dura and bony surfaces were then carefully curetted before the wound was closed.

The patient had an uneventful recovery, with gradual complete return of sensation and motor function of the lower extremities, and was discharged from hospital on November 16, 1927.

Pathology.—(M. G. H. S–27–876.) Microscopic examination (Fig. 10) shows a tumor made up of small round cells. In some areas these cells are enmeshed in a finely fibrillar supporting framework. Many blood capillaries are present. In other areas, the stroma is scanty, and tumor cells are loosely intermingled with a small number of branching reticular cells.

The capillaries are delicate, their walls consisting solely of a single layer of endothelial cells.

The tumor cells are small, but show some variation in size. (Fig. 11.) They are round, and contain only a small amount of cytoplasm. The nuclei are centrally placed, irregularly round, and deeply stained. Mitosis is rare. There are no eosinophils or fibroblasts.

Diagnosis.—Malignant lymphocytoma (lymphosarcoma).

Discussion.—The interest in this case centres upon the diagnosis of the tumor removed from the spinal canal. It is certainly not a primary extradural tumor, since lymphoid tissue does not occur in the spinal canal. If it is to be regarded as a metastatic tumor, its relationship to the tumors of the neck previously operated upon must be considered. In structure it is not absolutely identical with any of these, there being minor differences as regards stroma, vascular elements, and reticulum cells. The tumor cell in each case, however, presents the same characteristics. It is a small round cell with little cytoplasm, and a nucleus which is irregularly round and deeply staining.

Final Diagnosis.—A metastatic extradural malignant lymphocytoma of the spinal canal. The primary tumor was a malignant lymphocytoma of the cervical lymph glands first operated upon six years ago.

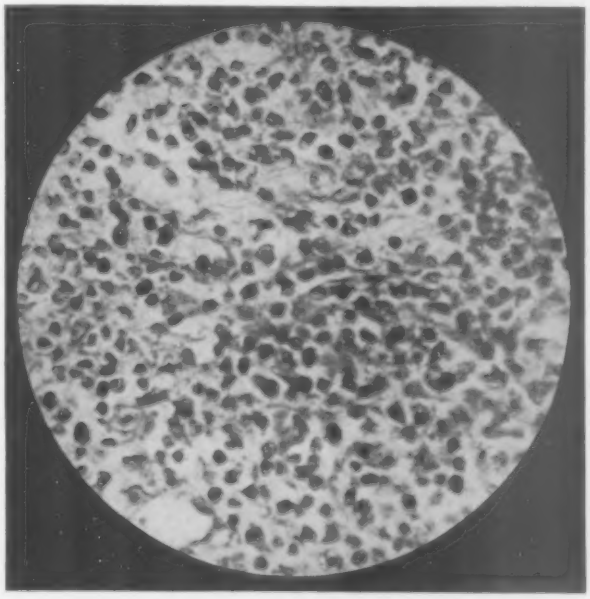


FIG. 11.—High power microphotograph of section from tumor of spinal canal removed in 1927, showing small round tumor cells supported by a fine fibrillar framework.

Metastases of lymphosarcoma within the spinal canal are extremely rare, and little reference to them is found in the literature. One case of primary intestinal lymphosarcoma which showed a secondary diffuse microscopical infiltration of the meninges, is reported by Sturzberg.[7]

Also of interest on account of the intraspinal metastasis, is a case reported by Welch,[8] of Hodgkin's Disease terminating in sarcoma. In this case there had been a paraplegia of many months' duration, and at autopsy a large metastatic extradural mass was found.

Extradural tumors produce changes in the cord by simple compression. At first, the cord is swollen and hyperæmic above the tumor, even though there is no actual destruction of nerve tissue. If the compression continues, however, destruction of the nerve tissue may result. The prognosis, following removal of the tumor, depends upon whether or not nerve tissue has been destroyed. If no actual destruction has occurred, functional recovery is rapid. Occasionally, an acute inflammatory process accompanies the tumor compression, and the prognosis in such cases is less favorable due to the more extensive degeneration of nerve tissue which results.

REFERENCES

[1] Ewing: Monograph on Neoplastic Diseases.
[2] Graves: Journal of Medical Research, September, 1919.
[3] Ochsner: Surgical Diagnosis and Treatment, 1921.
[4] De Noyelles: ANNALS OF SURGERY, August, 1922.
[5] Bloodgood: Progressive Medicine, vol. xxi.
[6] Minot and Isaacs: J. A. M. A., April, 1925.
[7] Sturzberg: *Ibid.*, pp. 33, 66.
[8] Welch: N. Y. Path. Soc., 1910, vol. x, p. 161.

RECURRENT FIBROMATOUS TUMORS OF THE SKIN

By David A. Willis, M.D.

of Chicago, Ill.

From the Department of Pathology, University of Illinois, College of Medicine

Contrary to the conventional conception of fibroma, viz.: a benign growth, which does not metastasize and which when enucleated does not recur, there is a fibromatous tumor of the skin which behaves otherwise and which possesses rather characteristic clinical and histological features.

In 1924, Darier [1] described these tumors under what he termed "Progressive and Recurring Dermato-fibromas." In the following year, Hoffman [2] reviewed the literature and reported three additional cases, naming the tumors "Dermato Fibrosarcoma Protuberance." Hertzler [3] in 1926 reported the very same type of tumor, but failed to associate the condition with that described by the previous writers. That recurrent fibromas are not necessarily confined only to the skin is illustrated by the case of Sichel [4] in which, following supravaginal hysterectomy for fibromas of the uterus, there occurred and recurred many times multiple free fibromas in the abdominal cavity, some quite far removed from the site of the original tumors. All the tumors were histologically cellular fibromas, each succeeding new growth being more cellular than the preceding one.

Very recently two cases identical with those in this series have been reported by Senear, Andrews and Willis,[5] which authors to our knowledge are the first to have associated these recurrent fibromas with the type of tumors described by Darier and by Hoffman.

From the few reports in the literature one might think the condition a rare one, but since we have, in a comparatively short time, collected four such cases, we feel that the condition is perhaps generally overlooked. Furthermore, because of the characteristic appearance of the tumors and the tendency to recurrence after removal, we have felt it important to call renewed attention to the condition and report our own cases.

Case I.*—Woman aged forty-five years gives the following history: Following a slight injury to the wrist twenty-four years ago, a small nodule appeared on the radial side of the wrist. The mass remained stationary and was removed about six years ago. One year later the tumor recurred, gradually enlarged and was again removed. The present tumor is another recurrence.

There is now present a central spherical tumor mass 3 cm. in diameter, projecting about 8 mm. above the skin surface, with scattered nodular tumors at its base ranging from 5 to 10 mm. in diameter. Over the larger mass, the skin is smooth and glistening and of a reddish-purplish color. The tumors are firm, definitely attached to the skin, but are freely movable over the underlying tissue.

On cut section the structure is uniform and pinkish-white with interlacing strands of more whitish bundles.

* Case I. Case of Dr. H. M. Richter.

CASE II.†—Woman fifty years of age was operated twelve years ago for a tumor mass in the inner portion of the left breast and also one on the back. About one year ago the tumors began to recur and in the last three months have grown rapidly. There is now present a tumor mass 3 x 5 cm. at the angle of the sternum and left breast. It is hard, freely movable, multilocular and not tender. No axillary glandular enlargements are palpable. A small nodule is also present on the back in the region of the left border of the twelfth dorsal vertebra.

CASE III.—No history could be obtained in this case but the clinical and histological appearance of the tumor warrants its inclusion in this series. The nodular elevations in this growth range from 1 to 5 cm. in diameter, are firm and whitish in appearance (Fig. 1), protrude about 1 cm. above the skin surface, and extend about 3 cm. below the skin surface.

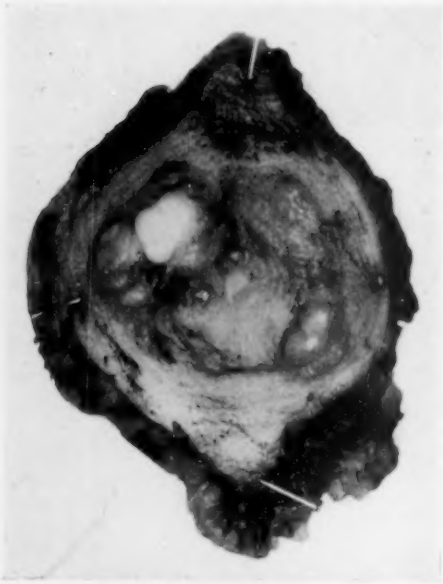


FIG. 1.—Showing the tumor nodules in the skin.

CASE IV.‡—Male patient about forty years of age had several tumors removed from the abdominal wall eight years ago. The growths have since recurred and there is now about ten protuberances on the abdominal wall, varying from 2 to 6 cm. in diameter, firm and whitish in appearance. Some are fairly well fixed, while the majority of the nodules are freely movable.

On cut section the surfaces appear to be composed of white fibrous-like tissue arranged in interlacing bands and whorls. No softening has occurred anywhere.

Histologically the tumors are composed of spindle-shaped cells in a dense fibrous stroma. In some places the connective tissue element is more abundant, while in others the cellular portion predominates. The nuclei of the cells possess a fairly heavy chromatin network and are somewhat more irregular than in the previous cases. Mitotic figures may be seen here and there. Lacunar-like blood-vessels are present.

The gross and microscopical appearances of all the tumors are so nearly alike that the following description will serve to illustrate the essential features in each.

Grossly the appearance of the nodular elevations varies with the duration and rapidity of the growth. The smaller and younger nodules have as a rule whitish tops, are only slightly elevated and the skin above is neither stretched nor glistening. The larger tumors may have whitish tops when the growth is rapid, but as a rule the color is a purplish-red, particularly in the very slow growing or quiescent elevations with secondary changes in their substance. The skin above is usually smooth and glistening. The individual nodules vary in size from a few millimetres to 4 cm. in diameter, the smallest being only palpable in the skin, but not visible. The elevation above the skin surface varies a great deal and there is no set rule as to this feature. Often they project as reddish spherical masses about 3 cm. above the skin surface. This, however, is no indication of the actual size of the tumor, for they usually extend much farther below than above the surface of the body. They are as a rule fixed to the skin above but are freely movable over the underlying structures. In consistency the younger and faster growing tumors are firm, while the older ones may be soft and even fluctuating.

† Case II. Case of Dr. Emil Beck.

‡ Case IV. Case of Dr. Edmund Andrews.

On cut section the structure is uniform and pinkish-white with interlacing strands of more whitish bundles. In the older modules, however, particularly those which are soft and fluctuating, there are often present small hemorrhagic areas and areas composed of a gelatinous-like substance.

Microscopically the tumors are composed of spindle-shaped cells with oval regular nuclei possessing a moderate chromatin network. The cells are generally uniform in size and shape, resembling typical fibroblasts, except in the long-standing quiescent nodules where the collogenous intercellular fibrillar tissue is markedly predominant and the individual cells are quite small but still spindle-shaped. Occasionally in such an old nodule an area of active cellular proliferation is present. Here the cells again predominate, are larger and again resemble fibroblasts. (Fig. 2.) They arrange themselves into distinct bundles with a varying amount of intercellular fibrillar tissue. The cells, however, are usually predominant. Mitotic figures are rare. The intercellular tissue shows little affinity to the acid stains. Even with Mallory's aniline-blue method it takes a light blue color, while with Van Giesen's stain the color is a pale dirty yellow. An occasional wide lacunar-like blood-vessel is present. (Fig. 3.) The skin above is not invaded but is stretched and flattened, the papillæ being entirely absent. Areas with mucoid degeneration and small hemorrhages are not infrequent in the old quiescent growths. In such areas the cells appear branched and are separated by a fluid which stains a pale blue with hæmatoxylin. Though a distinct capsule is absent, the tumors are well circumscribed from the surrounding tissue, this being due to an eccentric compression of the adjacent structures. At the base, however, there is a difference in the behavior of the tumor cells, for here they can be seen to break into the subcutaneous fat tissue forming finger-like projections between the fat cells. (Fig. 4.)

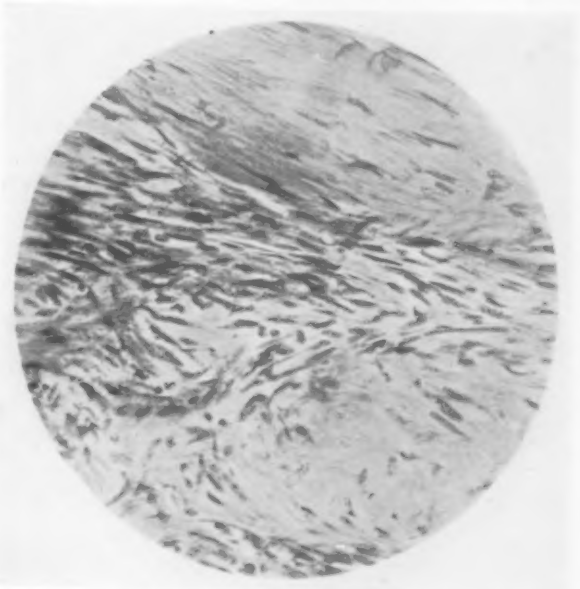


FIG. 2.—An area of cellular proliferation in an old tumor nodule.

Discussion.—It is quite apparent how the clinical course and histological appearance of the tumors might lead to a controversy between surgeon and pathologist, if one is not familiar with the condition. The tumors may occur anywhere on the body. There is usually a mother tumor larger than the surrounding elevations, which are apt to be arranged radially and which are often so small as to be hardly visible. The younger or more rapid growths have whitish tops, while the skin over the older ones are purplish-red in color. The masses are usually firm unless mucoid degeneration or hemorrhages in their substance have caused them to become soft and fluctuating. The cut surface is pinkish-white and glistening with prominent interlacing whitish strands. The tumor is attached firmly to the skin above, but can readily be

pulled away from the underlying fatty tissue. This gives the impression of the growth being encapsulated, but the encapsulation is more apparent than real, for as has been seen, the tumor cells actually grow into the subcutaneous fat in finger-like projections, differing in this respect from the common fibroma. It is evidently these invading portions which after operation account for the recurrences. There is no evidence of metastasis beyond the immediate region of the growth. Microscopically, the cellular element, the lacunar vessels and the staining qualities of the tissues are the characteristic features. Occasionally in a fine fibrillar form of tumor, the cells are arranged in pallisade formation. Lauche[6] and also Krumbein[7] have noted that such an arrangement is indicative of cellular proliferation of mesenchymatous origin, the arrangement according to Krumbein being found particularly in fine fibrillar fibromas, or as he calls them, "fibroma tennui—fibrillare." In this connection it is also interesting to note that Hertzler suggests that the tumors may represent sarcomatous growths derived from adult tissue.

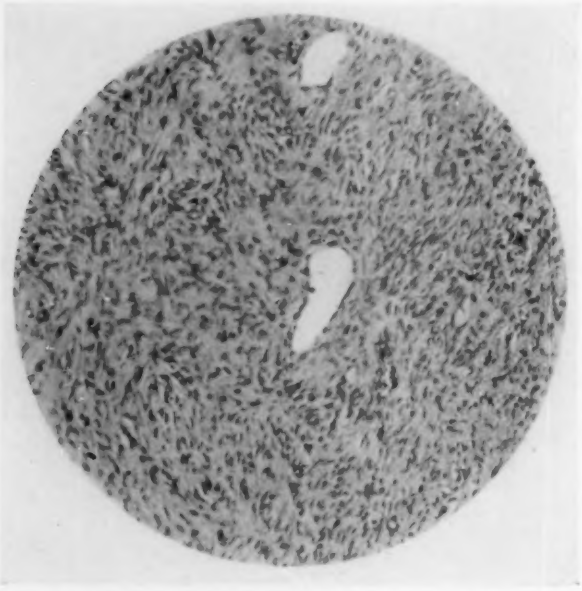


FIG. 3.—Lacunar blood-vessels in the substance of the tumor.

Treatment.—The clinical course and histological appearance of the tumors makes a radical removal imperative. As much as possible of the subcutaneous fatty tissue should be removed, the incision in the skin having circled at some distance beyond the edge of the nodules. Where thorough excision is not possible, radiation should be instituted, for since with each recurrence the tumor becomes more cellular, its eventual sarcomatous possibilities must be carefully considered.

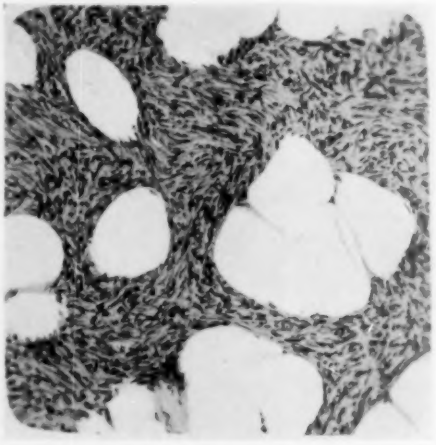


FIG. 4.—Tumor cells penetrating into the subcutaneous fat.

Conclusion.—(1) The fibromatous tumors of the skin here described no doubt belong to the same group described by Darier and by Hoffman.

(2) They possess a characteristic clinical and histological appearance.

(3) Radical excision is the only means of preventing recurrences.

REFERENCES

[1] Darier, J.: Dermatofibromes Progressifs et Recidivants. Ann. de Dermat. et Syph., vol. v, p. 545, 1924.

[2] Hoffman, E.: Uber das knollentreibende Fibrosarkoma der Haut. Dermat. Ztchr., vol. xliii, p. 1, 1925.

[3] Hertzler, A. E.: Fibrosarcomatous Tumors of the Skin of the Trunk. ANNALS OF SURGERY, vol. lxxxiv, p. 489, 1926.

[4] Sichel, M.: Mehrfach rezidivierde, multiple Fibrome in der freien Bauchhole. Zentralbl. f. Gynak., vol. l, p. 2363, 1926.

[5] Senear, F., Andrews, E., and Willis, D. A.: Dermato Fibrosarcoma Protuberans. Archives of Dermatology and Syphilology, 1928. In print.

[6] Lauche, A.: Uber rhythmische Strukturen in Menschlichen Geweben. Virchow's Archiv., vol. cclvii, p. 751, 1925.

[7] Krumbein, C.: Uber die Band-oder Pallisadenstellung der Kerne. Virchow's Archiv., vol. cclv, p. 309, 1926.

TRANSACTIONS

OF THE

NEW YORK SURGICAL SOCIETY

STATED MEETING HELD FEBRUARY 22, 1928

The President, Dr. Frank S. Mathews, in the Chair

POLYP OF DUODENUM

Dr. William B. Parsons, Jr., presented a man, colored, aged forty-one years, who was first seen at the Presbyterian Hospital, November 5, 1927, complaining of indigestion for four months and vomiting for six weeks.

For many years he had had chronic indigestion and he was chronically constipated for at least seven years. About six years before admission he had an acute episode of abdominal pain and a hemorrhage, at which time he had either vomited or had passed a large bloody stool. Following this attack he was well up to four months before admission. Four months before he came in he noticed that shortly after eating he had sour gaseous eructations accompanied by epigastric discomfort but not real pain. Under treatment he obtained some relief for a time but at the end of three months the previous symptoms returned, getting steadily more severe until just before admission to the Presbyterian Hospital, he had been vomiting immediately after taking fluids or solids and had also had definite gnawing pain brought on by food and relieved by vomiting. There had been no radiation of pain and no vomiting of blood before admission. On admission he weighed 109 pounds, was markedly emaciated and dehydrated. The positive physical findings in addition to his general appearance were in the abdomen where peristalsis was seen at various places. The margin of the liver could be seen about 3 cm. below the costal margin and a vague rounded mass was palpable near the pylorus. His blood was concentrated, the first and many subsequent stools showed a positive guaiac reaction. Gastric analysis showed a free hydrochlorate to 45, total of 61. A gastro-intestinal examination showed a marked pyloric spasm with active peristalsis above the spasm which did not succeed in moving the barium. Amyl nitrite was administered and almost immediately the active peristalsis ceased and the spasm definitely diminished. The pyloric portion then appeared as a narrow line curved to the right, down, backward, and then to the left but demonstration of the bulb was unsatisfactory. The second portion of the

Fig. 1.—Polyp of duodenum. Photograph of specimen.

duodenum was seen and it appeared to be dilated but there was difficulty in passing the barium along. After a month in the hospital his general condition had not improved. The vomiting had become more frequent and was on occasions projectile. Intestinal bleeding had continued, the pain had become more severe and more cramp-like in character. A proctoscopy was done. There was no evidence of colitis found but a single small polyp was removed. He was operated upon on December 13 with diagnosis of duodenal ulcer because of the spasm, pain, tarry stools and bloodless vomiting which seemed best explained on this basis.

At operation a polyp measuring 8½ x 5½ x 4½ cm. was found arising from a broad pedicle just behind the pyloric ring. The polyp arose from the posterior wall and had caused a marked dilatation of the first portion of the duodenum. The growth was composed almost entirely of mucous glands and was definitely arising from the mucosa. An anterior incision was made through the pylorus, the pedicle was excised and the wounds in the duodenal and gastric wall were closed.

The patient made a rapid recovery and is shown now, only two months post-operative, not because of any particular difficulties but because of the rarity of the condition.

Benign tumors of the small intestine are relatively infrequent, particularly those of the duodenum. Of the last 2300 autopsies at the Presbyterian Hospital, in three cases duodenal growths were found—in two of them a single mass and in the third several polyps associated with a general polyposis. In the last ten years there are fourteen cases on record in the Surgical Pathological Laboratory of benign tumors of the small intestine, two of these being polyps, one being the case reported and another one a case operated upon by Dr. A. O. Whipple, this polyp being composed almost entirely of Brunner's glands and was almost to be classified as an adenoma.

Dr. Ross Golden has recently reviewed the literature and will shortly publish in the *American Journal of Röntgenology* a report of Doctor Whipple's case together with the review of the literature. He quotes Doering's article in 1907 in which only three of fifty cases of polyposis of gastro-intestinal tract show involvement of duodenum; and also Versé who could find but one case in the large material in the Pathological Institute in Leipzig. The other types of tumor that Doctor Golden mentions in his article are myoma, adenoma and hemangioma.

Dr. William C. MacCarty (Rochester, Minn.) said he had seen two cases of polyp of the duodenum and both were resected. They had been reported by Doctor Pemberton. Neither was as large as the one reported by Doctor Parsons. He had never seen a malignant polyp in the duodenum. Polypi of the wall of the large bowel were more often malignant than they were believed to be a few years ago.

RESECTION. CARCINOMA OF THE STOMACH. RESULT FIVE YEARS AFTER

Dr. John E. Jennings presented a man, fifty-four years of age, who was admitted to the Brooklyn Hospital, March 12, 1923. Chief complaint, pain in epigastrium, digestive attacks for one and a half years. About one year

ago he began to have epigastric pain coming on one hour after eating. It was accompanied by nausea, gaseous eructations and vomiting of the previously eaten food. His sputum was negative. A test-meal showed free Hcl, 50, total acidity 80, blood and mucus plus, bile 0, urine acid 1010 and negative for albumin and sugar. The stomach showed at the angle of the lesser curvature a broad filling defect. In most of the films there is an incisura opposite this in the greater curvature. There was considerable gastric residue at five and a half hours. After twelve days of special diet and frequent feedings he was operated on and a mass the size of a walnut was found which involved the lesser curvature of the stomach, the posterior wall, the body of the pancreas and several lymph-nodes. There were several small nodules on the under surface of the liver. A gastrectomy was done using the posterior method of Polya. Examination showed the growth to be malignant, a carcinoma, apparently, on an ulcer base. The convalescence was uneventful and he has remained under observation as an employee of the hospital since his discharge from the ward. He is in good health without pain or dyspepsia. He has not gained much weight but is able to do his work which consists of running an elevator, without any distress. An X-ray of his stomach shows a very small and rapidly emptying organ. He is presented as a case of cancer of the stomach without recurrence five years after operation.

Dr. William C. MacCarty recalled a large carcinoma of the stomach which was removed in the Mayo Clinic five years ago; it was an enormous thing. When he opened the specimen he found two pieces of celery as large as his little finger. This patient is still alive and working.

SUPPURATIVE PERI-SIGMOIDITIS

Dr. John E. Jennings presented a man forty-nine years of age, who was admitted to the Brooklyn Hospital, November 11, 1927, suffering with acute abdominal pain, which had begun suddenly thirty hours before. It was generalized for several hours and then gradually became localized in the lower left quadrant. At the time of admission the abdomen showed well marked rigidity and tenderness in both lower quadrants, more pronounced on the left. The temperature was 101, pulse 120, respiration 24. Leucocyte count was 14,800—91 per cent. polymorphonuclear. An immediate laparotomy was performed. A frankly exploratory incision was made just to the right of the median line. A large quantity of foul, purulent fluid was found diffusely generalized. The small intestine was purple and distended. A leaking abscess was found at the pelvic brim on the left side surrounding a gangrenous epiploic appendix. The abscess cavity was opened and cleansed, packed lightly with iodoform gauze and the pelvis well drained with raffia drain tubes. He was placed in bed in the Fowler posture which calls for an elevation on the head of the bed thirty inches. He was given a hypodermoclysis of 1500 c.c. of saline containing 100 c.c. of perfringens antitoxin. Twelve hours later a second clysis was given of 1000 c.c. saline containing 100 c.c. of antitoxin. On the second day after operation he began to vomit stercoraceous material in spite of repeated gastric lavage which was done every four hours. His distention increased, his temperature persisted and his pulse remained about 120. His color, however, remained good and his pupils did not dilate. On the third day he was given 50 c.c. of a 20 per cent. solution of sodium chloride. He also was given eserine and pituitrin. His bowels moved several times and his distention diminished, vomiting ceased and temperature, pulse and respiration began to approach normal. But on the fifth day distention recurred and during a lavage the deeper layers of his wound opened and the intestine

appeared, only retained by the crossed tension sutures. They were promptly replaced by the house surgeon and iodoform gauze placed between the coils of the exposed bowel and the stay sutures. During the next three days his temperature rose to 103, the pulse to 130 with distention, vomiting and abdominal pain. He became irrational and the vomiting again became fecal. He was given 50 c.c. of a 20 per cent. solution of sodium chloride and 1000 c.c. with 200 c.c. of perfringens antitoxin by hypodermoclysis. His bowels moved, his vomiting ceased and he became rational in twenty-four hours. His temperature, pulse and respiration approached the normal. His bowels moved with a good deal of frequency during the next three days after which his convalescence was uninterrupted.

The use of Welch bacillus antitoxin in cases of gangrenous appendicitis with peritonitis was reported in 1922 after two years' experience, and its use as prophylactic in wounds of the colon and operations upon that organ, in rectal surgery and in fecal infection in general was reported. It has since that time been used in France and England where more sera are available than we have at present at our command.

The clinical study of abdominal infection due to anaërobic invasion from the gastro-intestinal tract has been singularly neglected and deserves attention. He hoped to discuss his own experiences more fully at another time. The use of hypertonic salt solution intravenously in cases of ileus was suggested and used by Hayden and Orr following the original studies of Hartwell and Hoguet. This case was presented in order to illustrate the very definite use, in certain cases of fecal infection, of an antitoxin prepared against the Welch bacillus and also to report a rather striking instance of an intravenous injection of hypo-tonic salt in intestinal obstruction. He had been lead to begin the use of Welch antitoxin by noting the presence of the Welch bacillus in infections of the abdominal wall following gangrenous appendicitis. Study showed that a fair proportion of fatal cases of appendicular peritonitis revealed the presence of the Welch bacillus in large numbers. It was found that Welch antitoxin in liberal doses was useful.

The experiments of Hayden and Orr suggest that the intoxication of intestinal obstruction is produced by a substance which affects the sodium chloride of the blood stream, absorbing the chlorine, leaving the sodium to circulate as sodium carbonate. This will account for the diminished blood chloride and if it be true, implies that salt is a chemical antitoxin. Clinically it appears to be so. As to the question of why it makes the bowels move, the reply is that probably this is due to the restoration of chloride tension in the tissue where its presence is most keenly missed. Clinically and experimentally intravenous injections of strong solutions of sodium chloride will cause contraction of the obstructed gut. It is safe to use one gram to the kilo of body weight.

SYMPATHECTOMY FOR THROMBO-ANGIITIS OBLITERANS

Dr. John E. Jennings presented four cases of threatened gangrene of the foot with several methods of treatment and varying results.

Case I.—A man, forty-three years of age. Very heavy cigarette smoker. Ten years ago began to have soreness of thumb of left hand. Later toes of

both feet were amputated and left foot also. Present attack eight months ago when he thought his foot had been frost bitten. The first, second and third toes of the right foot were gangrenous. The left leg had been amputated in its lower third. There is an ulcer at its lower extremity.

Operation.—Sympathectomy. Incision along the anterior border of the sartorius from a point opposite the upper edge of the patella six inches upward. Muscles retracted, vessel sheath exposed. Many evidences of peri-arteritis and peri-phlebitis made isolation of artery difficult. Artery was without pulsation. The artery and veins were separated and faint pulsation returned in artery which again ceased as soon as dissection of the wall began. The adventitia was stripped for a distance of three inches. Second and third toes and first phalanx of first as well as ulcer of the stump removed with the high frequency cutting current. Very slow and painful healing. Gangrene arrested. Pain not well controlled.

Case II.—A. M., Russian Jew, thirty-six years of age, February 7, 1927. Pain in great toe of right foot for four and a half months. Most pain in foot but also in ankle and up to the knee. The toe is white at the tip and purple at the base. The dorsalis pedis, posterior tibial, popliteal arteries cannot be felt in either foot. Sympathectomy with same technic as that just described. Pain was relieved for a short while only. Phlebitis of right femoral vein. Much relieved by hypertonic salt injections and by cutting off cigarettes. Now in a period of remission.

Case III.—L. E., born in United States of German Christian parents, fifty-three years of age. Six years ago cramps in both feet. Treated for flat feet. Agonizing pain, burning in character in the great toe of the left foot. Amputation of toe three months ago, ulcerating area still very painful. Is an inveterate smoker. *Operation.*—Ligation of femoral artery below the profunda. Immediate gangrene of leg extending to the knee. Amputation in the middle third of the thigh. Guilotine. Subsequent plastic on stump with sloughing of skin flap. Still granulating and almost healed.

Case IV.—Threatened gangrene of left great toe. Thrombo-angiitis obliterans. I. B., Hebrew, twenty-five years of age. During past winter left great toe was frozen. For past three months has had pain in toe and in the dorsum of the foot. Five weeks ago a small reddened area on the mesial aspect of the distal phalanx of the left great toe broke down and began to slough. There is now an area of painful ulceration. Previous history negative. Is a confirmed smoker of Turkish cigarettes. No pulsation in left dorsalis pedis or posterior tibial. *Operation.*—As in cases previously described. Opening Hunter's canal. Three inch sympathectomy of femoral artery. Relief of pain and healing of ulcer.

In the first case an advancing gangrene of the toes was arrested with slow but complete relief of pain.

In the second case a slowly progressing gangrene was arrested. The pain was not relieved till the hypertonic salt solution was administered, after which pain ceased.

In the third case ligation of the femoral as suggested by Lewis was practiced with immediate loss of the extremity.

In the fourth case, an early case with threatened gangrene, complete arrest of symptoms.

Dr. Walter A. Sherwood remarked that the operation of peri-arterial sympathectomy had fallen into a certain amount of disrepute. He felt, how-

ever, that this was due to the misapplication of the operation in unsuitable cases, and that nothing more than palliation or relief of pain should be expected in cases with obstructive lesions of the blood-vessels. In cases, however, in which spasm of the vessels is the underlying factor, as in Raynaud's disease, painful ulcers of amputation stumps and similar conditions, the operation has a distinct field of usefulness and has proved of value in numerous instances. The patient shown here several years ago by Doctor Sherwood, and who had suffered for over twenty years with a painful ulcer of the amputation stump, has remained entirely well since he was subjected to the Leriche operation. In arteriosclerotic gangrene or in thrombo-angiitis obliterans one should not expect anything more than temporary relief of pain.

Doctor Jennings in closing the discussion said that the operation performed on the four patients shown tonight was more than peri-arterial sympathectomy. Hunter's canal was widely opened in all the cases and a sufficient degree of peri-vascular tension was found to make relief of obstructive pressure on the vessels an important indicator of treatment. It appeared to him that this factor was of some importance. It seemed to him that there were two sorts of pain occurring in these cases; that one was due to spasm of the larger arteries and temporary relief might be expected in these cases as a result of sympathectomy. This pain is paroxysmal, much affected by activity, little by position and hardly at all by temperature changes. The other variety of pain resembles a terminal neuritis, which it probably is. It is much more constant, much more affected by temperature, posture or pressure and is little affected by a sympathectomy. In spite of Berger's brilliant contribution the disease does not apparently always follow the course described by him and thrombo-angiitis obliterans is only one of the pathological changes the essential nature of which is not as yet at all understood.

ACUTE PERFORATION OF STOMACH; SUTURE; RECURRENT SYMPTOMS; RESECTION

Dr. Richard Lewisohn presented a man, twenty-nine years old, who had been admitted to Mount Sinai Hospital, April 23, 1926, with a history of gastric symptoms for four weeks. Four hours previous to his admission he was suddenly seized with severe cramps in the abdomen, radiating to the right shoulder. He was nauseated but did not vomit. There was a general rigidity of the whole abdomen. Temperature was 100°, pulse 108. Immediate operation was performed by Doctor Stenbuck through a right upper rectus incision. A large amount of pus was found in the upper abdomen with a pyloric perforation. The perforation was closed and the abdomen drained by a rubber dam. Closure of the abdominal wall in layers. He made an uneventful recovery and left the hospital May 7, 1926.

The man was re-admitted, August 22, 1927, having been well for one year following his operation. During the last few months he had complained of heartburn and hunger pains two hours after the meal. X-ray examination showed a deformity of the bulb and reversed peristalsis in the second and third portions of the duodenum. A small residue was noticed on the posterior wall of the duodenum (niche). There was a slight residue in the stomach after six hours. Pre-operative Rehfus test-meal showed free acid 74, combined acids 90.

September 3, 1927 (Lewisohn). Under spinal anæsthesia a midline incision was made from the ensiform process to the umbilicus. Many firm adhesions were found between the omentum and the anterior abdominal wall and between the stomach and the liver and gall-bladder. A puckered scar was noticed at the pylorus (site of the old ulcer). In the posterior wall of the first part of the duodenum a firm, indurated ulcer was felt, adherent to the head of the pancreas and perforating into it. A typical Billroth II resection with a Hofmeister anastomosis was performed. The specimen showed that the ulcer in the first part of the duodenum had perforated into the pancreas and that the head of the pancreas formed the base of the ulcer. Closure of abdomen without drainage. The specimen in addition to the ulcers mentioned above showed a marked gastritis. The patient made an uneventful recovery and was discharged October 1, 1927.

Reëxamination, February 1, 1928: Patient is in perfect condition. Rehfus test-meal shows free acid 0, total acidity 12.

LATE RESULTS IN PERFORATED GASTRO-DUODENAL ULCERS

Dr. Richard Lewisohn read a paper with the above title for which see page 855.

Dr. Fordyce B. St. John emphasized the error of the old tradition, namely, that an acute perforation of an ulcer, simply treated, necessarily leads to a complete healing. Although follow-up clinics have made this apparent during the past ten years, it is worthy of notice that Doctor Lewisohn's figures show a majority of cases (in a ratio of 20 to 13) relieved of all symptoms.

Before the detailed study of the 39 per cent. "failures," of the thirty-three cases followed, the fact is mentioned that twenty, or approximately 61 per cent., are symptom-free. One must appreciate the importance to the patient of a simple procedure such as was carried out in these cases, from the standpoint of saving life, which after all is the primary object in such an operation at the time it is performed. He did not believe that any experienced surgeon feels that he is doing more than a life-saving procedure, in the presence of a grave complication in a disease the cause of which is unknown, and the treatment of which so debatable.

Doctor Lewisohn has referred in passing to the 34 per cent. of marginal ulcers following gastro-enterostomy at the Mt. Sinai Clinic, and suggests as an explanation that the high percentage is probably due to the fact that the cases are so thoroughly followed. Inasmuch as other follow-up Clinics in which cases are very carefully studied do not show the same high percentage of marginal ulcer, it did not seem to him that this explanation was satisfactory.

The Mt. Sinai Clinic is much the same as the Presbyterian Hospital Surgical Clinic as regards follow-up results in perforated ulcer, as is shown by Doctor Lewisohn's figures. In fifty cases at the Presbyterian during approximately the same period, there were seventeen deaths (two of which were before operation) or 30 per cent. following operation, and of the thirty-three living cases, twenty-seven cases, or 90 per cent., have been carefully followed, the records over a period of from two to ten years showing sixteen cases, or 59 per cent., to be symptom-free.

As regards the first of the two main points of Doctor Lewisohn's paper, it must be added that probably the lives of 70 per cent. of these patients were saved by the simple procedure carried out—a point which certainly is not to be minimized.

As regards the second point, that is, the very high percentage of multiple ulcers, the Presbyterian Hospital experience does not corroborate these findings.

As to the conclusions—he did not believe it possible to perform a subtotal gastrectomy in an acute perforated gastric or duodenal ulcer of from six to twelve hours' duration, without incurring a definite risk of mortality. And though he quotes continental observers as advocating this procedure, it seems to him that Doctor Lewisohn is wise in adding, "we believe the mortality would be materially increased", as otherwise one must disregard one's conceptions of the effect of trauma, exposure, duration and extent of surgical procedure as causes of shock.

It would seem to him sounder judgment carefully to follow these cases, and in the event of evidence of recurrence, to then decide on what further operative therapy should be carried out.

Dr. Charles E. Farr gave a summary of 117 operations for acute perforations of stomach and duodenum performed in the First Surgical Division (Cornell) of the New York Hospital.

No. of cases	No. hours	Deaths	Per cent. deaths
90	12 hrs.	11	11.7% (9.5%)
4	18		
9	24 hrs.	2	22.2%
14	Over 24 hrs.	8	57.1%

Operative mortality 17.9%

Type of operation	No. cases	Deaths	2nd operation	3rd operation
Suture of perforation	83	18	7* 2† 2‡	2§
Suture and appendectomy	21	0	5*	0
Suture and gastro-enterostomy	6	1	1*	0
Suture and herniotomy	1	0	1*	0
Suture and cholecystectomy	3	0	1*	0
Suture and drainage of gall-bladder	1	0	0	
Gastric resection	1	1	0	
Gastro-enterostomy only (Perforation could not be reached)	1	1	0	

* Gastro-enterostomy.
† Suture of second perforation.
‡ Resection.
§ Freeing of adhesion.

Results.—Twenty-one deaths following operation for acute perforation.

Of the remaining ninety-six cases:

2 were never seen after discharge.
70 had a good result after first operation.
2 cases are too early to include in statistics.
2 unsatisfactory.
20 cases required a second operation.

Final results:—Gastro-enterostomy (sixteen cases).—One case died a year after operation, of pulmonary tuberculosis. No further trouble with stomach. Two cases required a third operation for adhesions. Of these one died after third operation—intestinal obstruction by adhesions. The other case has an excellent result. Thirteen cases have a good result.

Suturing of second perforation (two cases). One case died after second operation (included in twenty-one deaths). One case satisfactory.

Gastric resection (two cases). One case satisfactory. One died after second operation.

Dr. John Douglas said that mortality statistics of operations for perforated ulcer are so dependent on the length of time between the perforation and the operation that it is unfair to compare these statistics according to methods.

One thing which had not been considered here this evening, not without bearing on the subject, is that a considerable number of these pyloric ulcers are so close to the pyloric ring that it is difficult to say whether they are gastric or duodenal. In a large indurated ulcer which has perforated, when the sutures are put in through inflamed tissue they sometimes cut through. If the ulcer is excised this, of course, lessens this danger and makes the closure both safer and simpler. If the perforation is near the pyloric ring and a pyloroplasty is done, after excising the ulcer, the pyloric opening is enlarged and the chances of recurrence of symptoms are less. Of course, it must be acknowledged that there are recurrences of ulcer symptoms in a considerable number of cases where a simple closure is done and there are some recurrences of symptoms, although less, if an excision of the ulcer and a pyloroplasty are employed. However, reports have been made of symptoms recurring after a subtotal gastrectomy and two cases have been reported from the Mayo Clinic of renewed ulceration after such an operation. Before accepting this radical operation as a positive cure surgeons should wait a little while longer.

Doctor Lewisohn, closing the discussion, said that he did not advocate subtotal gastrectomy in acute perforated gastric or duodenal ulcer. What he had wanted to point out was that these cases should be carefully followed and one should not feel when they were sent home that they were cured. A large number recur; if they are not relieved of their symptoms in six months they should be subjected to more radical procedures.

BRIEF COMMUNICATION

CRITIQUE ON ADRENALECTOMY FOR ALLEGED HYPERADRENALINEMIA

CONCERNING the report by the Russian surgeon, Oppel, entitled "Adrenalectomy for Hyperadrenalinemia in Spontaneous Gangrene," appearing in this number of the ANNALS OF SURGERY (vol. lxxxvii, No. 6, June, 1928, p. 801), which has been referred to us for criticism, we feel that all reliable experimental evidence, at present available, must lead one to condemn such a procedure on human beings. The proof accepted by Oppel and his collaborators, to support the idea that certain clinical conditions are associated with an excess of circulating adrenalin, is inadequate and the conclusions unwarranted. We base our criticism chiefly on the evidence afforded in the recently published paper by one of his assistants, Achutin, in the *Ztschr. f. d. ges. exp. Med.* (1927, vol. lvi, p. 698). From this article it appears that these investigators ignore, or are apparently not familiar with the *quantitative* studies, made by *quantitative* methods, on the rate of adrenalin discharge from the adrenal glands. In reaching their conclusions, they have employed the reactions of the isolated strip of cat's intestine to adrenalin.

The sympathomimetic effect, of adrenalin in effective concentrations, (*i.e.*, the same effect as is produced by stimulation of sympathetic nerve supply) on an intestine segment is inhibition of contractions and diminution of tone. This reaction is useful in making studies on the amount of adrenalin in blood and the reaction, when properly employed, yields satisfactory information concerning the concentration of adrenalin present. But from the above-mentioned article it appears that the writer has accepted rather crude *qualitative* reactions as a basis for *quantitative* interpretations. Reference to any of the large number of papers by Stewart and Rogoff, on quantitative measurements of adrenalin secretion under various conditions, will show that Achutin does not apply the method properly for quantitative studies. We have found the isolated rabbit's intestine segment more satisfactory than cat's and for very low concentrations of adrenalin in blood, the reaction, when doubtful, is always confirmed by utilizing a segment of rabbit's uterus (non-pregnant) which yields an effect opposite to that obtained on the intestine (*i.e.*, increase of tone and contractions). By the use of isolated segments of these two organs we have detected, with certainty, concentrations of adrenalin as low as 1:100,000,000 to 1:1,000,000,000 in blood or serum.

In a number of attempts made by us to detect adrenalin in the systemic blood, we have been unable, thus far, to obtain any definite reaction. This is not surprising since the epinephrin-containing blood from the suprarenal vein is diluted at least 200 times before reaching the general circulation and even on

the assumption that none of the epinephrin is destroyed in the pulmonary circulation (an assumption that is by no means justifiable) the concentration of adrenalin in systemic blood, under normal conditions, would probably be at most 1:1,000,000,000 to 1:2,000,000,000. However, in adrenal vein blood, where the concentration is within the limits capable of detection by our method, we have been able to make extensive quantitative studies on the rate of secretion.

From very inadequate tests, these Russian writers have been led to assume the existence of hyperadrenalinemia in such conditions as Raynaud's disease and spontaneous gangrene, and on the strength of what we feel are erroneous interpretations, have repeatedly operated upon human beings and removed one adrenal. Even if it were possible to consider the evidence offered by Achutin as valid and all the reactions observed by him as genuine adrenalin reactions, we feel that his conclusions are not warranted, owing to apparent lack of critical analysis of the alleged results. Since, if the reactions are interpreted as evidence of such concentrations of adrenalin in the systemic blood as he believes to exist, a simple arithmetical calculation would, at once, show that this is not only quite improbable but perhaps impossible. For, it would mean that the adrenalin concentration in the adrenal vein blood must be about 1:100,000 to 1:250,000. From the large number of quantitative studies made by us on the rate of adrenalin secretion from the adrenal glands, we consider the existence of such concentrations of adrenalin in the adrenal vein blood as highly incredible. Blood containing such concentrations of adrenalin being constantly discharged into the circulation, at the rate of blood flow that exists in the adrenal glands, would unquestionably produce tremendous reactions and would probably soon lead to a fatal outcome. It must be repeated that, in our opinion, excision of adrenals, in human beings, is too dangerous a procedure to undertake on the strength of these investigations, and should be deprecated.

J. M. ROGOFF, M.D.,

The H. K. Cushing Laboratory of Experimental Medicine, Western Reserve University, Cleveland, Ohio.

EDITORIAL ADDRESS

The office of the Editor of the Annals of Surgery is located at 489 Washington Avenue, Brooklyn, New York. All contributions for publication, Books for Review, and Exchanges should be sent to this address.

Remittances for Subscriptions and Advertising and all business communications should be addressed to the

ANNALS OF SURGERY
227-231 South Sixth Street
Philadelphia, Penna.

INDEX TO VOLUME LXXXVII

A

Abdominal Incisions, 74.
Abdominal Injuries in Children, 718.
Abdominal Wall, Progressive Gangrenous Ulceration of, 245.
Abduction Treatment for Fracture at Hip-joint, 442.
ABELL, IRVIN: Primary Hypernephroma of the Liver, 829.
Abscess, Acute Subpectoral, 517; Lung, Following Pneumococcus Septicæmia, 141; Perinephritic, 301; Post-pneumonic Lung Rupturing into Pleura, 624; Posterior Mediastinal, 511.
Adrenalectomy for Hyperadrenalinemia in Spontaneous Gangrene, 801; Critique on, 959.
ALBEE, FRED H.: Formation of Radius Congenitally Absent, 105.
Anæmia, Pernicious, Splenectomy for, 314.
Anæsthesia, Brachial Plexus, 883; Colonic, in Operations Upon Brain, 161.
Anæsthetics and Narcotics, Untoward Effects of, 124.
Anastomosis, Intestinal, Aseptic End-to-end, 861.
Aneurism, Ruptured Popliteal, 455.
Ankylosis of Knee, 609.
Anomalies, Branchial, and Neoplasms, 186.
Appendicectomy, Multiple Complications Following, 461.
Arterial Embolectomy, 667.
Arteriorrhaphy, Resection and Reconstructive, 462.
Arteriovenous Fistula, Acquired, 19.
ASHHURST, A. P. C.: Splenectomy for Blood Dyscrasia, 130.

B

Bastianelli: Testimonial to Dr. Raffaele, by His Pupils and Friends. Review of, 160.
BEEKMAN, FENWICK: Head Injuries in Children, 355; Intracapsular Fracture of Capitellum of the Humerus, 789; Plastic for Ulcer of the Leg, 790; Tannic Acid Treatment of Burns, 292.
BEER, EDWIN: Operative Removal of Kidney Stones, 428.
BELL, LEWIS BARCLAY: Primary Sarcoma of the Clavicle, 382.
BIGELOW, LESLIE L.: Lymphosarcoma of the Mesentery, 879.
Biliary Surgery, Remote Results of, 837.
Bile Duct, Cystic Congenital Dilatation of Common, 48; Effects of Obstruction of the, 210.
Bladder, Urinary Diverticula of the, 742; Intraperitoneal Rupture of, During Abdominal Examination, 465; Traumatic Rupture of, in Children, 307.
Blood Dyscrasia, Splenectomy for, 130.
Blood Transfusion, The Technic of, 775.
Bloods, Laked and Traumatized, and Blood from Traumatized Limbs, 806.
Bones, Hodgkin's Disease of, 755.
Bowel and Iliac Vein, Bullet Wounds of, 132.
Bowel, Rupture of, at Duodeno-jejunal Junction, 131.
BOYKIN, IRVINE M.: Abdominal Incisions, 74.
Brachial Plexus Anæsthesia, 883.
Brain, Colonic Anæsthesia in Operations upon, 161; Spongioblastoma Multiforme of the, 8.
Brain-tumors, Removal of, 779.
BRANCH, J. R. B.: Enterostomy in Typhoid Perforation, 476.
Branchial Anomalies and Neoplasms, 186.
BRILL, SELLING: Splenectomy for Pernicious Anæmia, 314.
Bronchiectasis, Lobectomy for, 607; Unilateral, 134.
BROWN, HENRY P., JR.: Surgical Intervention in Extra-uterine Pregnancy, 581.
BROWN, JAMES BARRETT: Sarcoma of Ovary with Unusual Oral Metastases, 467.
BRUGGEMAN, HENRY OTTO: Treatment of Acute Cholecystitis, 423.
BUCHMAN, JOSEPH: Traumatic Osteoporosis of the Carpal Bones, 892.
Burns of the Eye, Acid and Alkali, 89.
Burns, Tannic Acid Treatment of, 292.
Buttock, Melanoma of, 621.

C

Carcinoma of Chest Wall, Recurrent, 605; of the Face, Relief of Pain in, 179; Plastic, of Face and Jaw Following Excision for, 316; of the Ovary in Infancy, 84; Primary, of the Hepatic Duct, 157; of the Rectum, 130, 790; of the Stomach, 951.
CARP, LOUIS: Branchial Anomalies and Neoplasms, 186.
Carpal Bones, Traumatic Osteoporosis of the, 892.
CARSON, WILLIAM JAMES: Solitary Cysts of the Kidney, 250.
Cautery-pneumectomy, 159.
Chancre Complicating Laceration of Hand, 783.
Chest, Visor Flaps from the, 364.
Chest Wall, Recurrent Carcinoma of, 605.
CHETWOOD, CHARLES H.: Practice of Urology and Syphilology. Edited by. Review of, 478.
Children, Abdominal Injuries in, 718; Head Injuries in, 355.
Cholangitis Following Cholecystenterostomy, 54; Cholecystectomy, Gastroenterostomy Following Adhesions After, 300.
Cholecystenterostomy, Cholangitis Following, 54.
Cholecystenterostomy for Chronic Pancreatitis, 300.
Cholecystitis, Treatment of Acute, 423.
Cholecystogastrostomy, 146.
Choledochoduodenostomy, 784.
Cholelithiasis and Large Umbilical Hernia, 299.
Chromoma of the Forearm, 99.
CLARK, JOHN G.: Radium in Gynæcology, Treatise on. Edited by. Review of, 479.
Clavicle, Primary Sarcoma of the, 382.
Cleft Palate and Harelip, 536.
Colitis, Ulcerative, 612; Ileostomy for, 799.
Collapse, Massive, of Right Lung After Nephrectomy, 600.
Colon, Gangrene of Transverse, Strangulated Umbilical Hernia with, 144.
Colonic Anæsthesia in Operations upon Brain, 161.
Colostomy, A New Type of Permanent, 711.
Common Duct, Repair of, 784.
Compound Injuries of Extremities, Treatment of, 321.
CONNELL, F. GREGORY: Remote Results of Biliary Surgery, 837.
Coronary Disease in Surgical Patients, 32.
COSGROVE, KINGSLEY W.: Acid and Alkali Burns of the Eye, 89.
COSTELLO, CLARENCE V.: Sublingual Phlegmon, 684.
COUNSELLER, VIRGIL SHEETS: Effects of Obstruction of the Bile Ducts, 210.
Cranial Decompressive Operations, Dura Mater in, 15.
Cranial Meningocele and Spina Bifida, 811.
Cyst, Infected Congenital, of Lung, 606; Ovarian Rupture of, 307.
Cystic Dilatation, Congenital, of Common Bile Duct, 48.
Cystoscopy Reactions, 435.
Cysts, Kidney, Solitary of the, 250; Liver, Non-parasitic of the, 844.

D

DANZIS, MAX: Arterial Embolectomy, 667.
DARNALL, WILLIAM EDGAR: Fibromata of the Mesentery, 870.
DAVID, VERNON C.: Pseudo-carcinoma of the Stomach, 555.
DAVIS, BYRON B.: Mortality and End Results in Gall Bladder Surgery, 735.
DAVIS, LOYAL: Spongioblastoma Multiforme of the Brain, 8.
DAVIS, WARREN B.: Harelip and Cleft Palate, 536.
DECOURCY, JOSEPH L.: Treatment of Ulcer of the Stomach and Duodenum, 566.
De Petz: Stomach and Intestinal Suturing Apparatus, 80.
Diabetes and Hyperthyroidism, 37.
Diverticula of the Urinary Bladder, 742.
DIXON, CLAUDE F.: A New Type of Permanent Colostomy, 711.
DOUGLAS, JOHN: Resection of Superior Maxilla for Osteosarcoma, 303.
Duct, Repair of Common, 784.
Duodenal and Gastric Ulcers, Concomitant, Two-and-one-half Years Post-operative, 143.
Duodenal Ulcer, Perforating, 292.
Duodeno-jejunal Junction, Rupture of Bowel at, 131.
Duodenum and Stomach, Ulcer of the, Treatment of, 566.
Duodenum, Polyp of, 950.
Dura Mater in Cranial Decompressive Operations, 15.

E

Eggers, Carl: Surgical Treatment of Suppuration of the Lung, 485.
Eliason, E. L.: Acute Sigmoiditis, 783; Bullet Wounds of Bowel and Iliac Vein, 132; Choledochoduodenostomy, 784; Repair of Common Duct, 784; Rupture of Bowel at Duodeno-jejunal Junction, 131.
Elsberg, Charles A.: The Rôle of the Dura Mater in Cranial Decompressive Operations, 15.
Embolectomy, 652; Arterial, 667.
Embolism, Pulmonary, 528.
Empyema Following Pñeumothorax Therapy for Tuberculosis, 604; Massive, Obliterative Operations for, 506.
Enterocystoma, Ileocæcal, Producing Partial Intussusception, 676.
Enterolith Suggesting Vesical Calculus, 154.
Enterostomy in Typhoid Perforation, 476.
Epinephrectomy for Hyperadrenalinemia in Spontaneous Gangrene, 801.
Extra-uterine Pregnancy, Surgical Intervention in, 581.
Extremities, Puerperal Gangrene of the, 767; Lower, Thrombophlebitis of the, 641; Treatment of Compound Injuries of, 321.
Eye, Acid and Alkali Burns of the, 89.

F

Face and Jaw, Plastic of, Following Excision for Carcinoma, 316.
Face, Relief of Pain in Carcinoma of the, 179.
Failla, Gioacchino: Radium in Gynæcology. Edited by. Review of, 479.
Femur, Fracture of Neck of, 787; Abduction Treatment for, 320.
Ferguson, L. K.: Acute Suppurative Pancreatitis, 133.
Fibroma of Lower Jaw, 305.
Fibromata of the Mesentery, 870.
Fibro-sarcoma of Neck, 621.
Fisher, William H.: Lymphangioma of Omentum, 872.
Fistula, Acquired Arteriovenous, 19; Gastro-jejuno-colic, 578.
Fistulæ, Gastro-jejuno-colic, and Gastro-jejunal Ulcers, 231.
Flick, John B.: Unilateral Bronchiectasis, 134.
Forearm, Chromoma of the, 99.
Fractures, French-Heel, of Tarsal Scaphoid, 587; of Hip, Flexed Plaster Spica Case for, 111; of Neck of Femur, Abduction Treatment for, 320.
Frazier, Charles H.: Colonic Anæsthesia in Operations upon Brain, 161.
Freeman, Leonard: Visor Flaps from the Chest, 364.

G

Gall Bladder Surgery, Mortality and End Results in, 735.
Gangrene, Gas, The Prophylaxis of, in Civil Surgery, 772.
Gangrene, Puerperal, of the Extremities, 767; Spontaneous, Epinephrectomy (Adrenalectomy) for Hyperadrenalinemia in, 801.
Garlock, John H.: Treatment of Compound Injuries of Extremities, 321.
Gas Gangrene in Civil Surgery, The Prophylaxis of, 772.
Gastric Ulcer, Accessory Pancreas Simulating, 150.
Gastric and Duodenal Ulcers, Concomitant, Two and one-half Years Post-operative, 143.
Gastritis, Phlegmonous, 848.
Gastro-duodenal Ulcers, Late Results in Perforated, 855.
Gastroenterostomy Following Adhesions After Cholecystectomy, 300.
Gastro-jejunal Ulcers and Gastro-jejuno-colic Fistulæ, 231.
Gastro-jejuno-colic Fistula, 578.
Gastrostomy, Prophylactic, 395.
General Practice, Exigencies of a, by Nathan Clark Morse, Review of, 640.
Gerster, John C. A.: Suspension of the Foot in Pott's Fracture, 450.
Gibbon, Robert L.: Acute Intussusception in Infants, 318.
Gibson, Charles L.: Cholecystenterostomy for Chronic Pancreatitis, 300; Gastroenterostomy Following Adhesions After Cholecystectomy, 300; Perinephritic Abscess, 301; Polya-Mayo Exclusion for Duodenal Ulcer, 301.
Goitre, Congenital, 297.
Grant, Francis C.: Relief of Pain in Carcinoma of the Face, 179; Removal of Brain Tumors, 779.
Grausman, Philip M.: Cystic Lymphangioma of the Greater Omentum, 66.
Graves' Disease in the Male, 296.

GREENE, LLOYD B.: Traumatic Rupture of Urinary Bladder in Children, 307.

H

Hæmolytic Icterus—Splenectomy, 459.
Hæmophilia in an Adult, 593.
HALL, LYMAN S.: Pulmonary Embolism, 528.
Hand, Chancre Complicating Laceration of, 783.
HANFORD, JOHN M.: Anomalous Hepatic Duct, Pericholecystitis, 151.
Harelip and Cleft Palate, 536.
HAUSER, GEORGE H.: Puerperal Gangrene of the Extremities, 767.
Head Injuries in Children, 355.
Heart Injuries with Suture, 823.
HEGNER, CASPER F.: Obliterative Operations for Massive Empyema, 506.
HENDERSON, MELVIN S.: Derangements of the Knee-joint, 911.
Hepatic Degeneration, Acute, 146.
Hepatic Duct, Anomalous, Pericholecystitis, 151; Primary Carcinoma of the, 157.
HERMAN, LEON: Massive Collapse of Right Lung After Nephrectomy, 600.
Hernia into Lesser Peritoneal Cavity, 312; Strangulated, Reduction "En Bloc" of, 313; Strangulated Umbilical, with Gangrene of Transverse Colon, 144; Unilateral, Transverse Incision for, 639.
HERTZLER, ARTHUR E.: Chromoma of the Forearm, 99.
HEYD, CHARLES GORDON: Accessory Pancreas Simulating Gastric Ulcer, 150; Acute Hepatic Degeneration, Cholecystogastrostomy, 146.
HIGGINS, CHARLES CLAIR: Malignant Tumors of the Testicle, 263.
HINTON, J. WILLIAM: Chronic Pancreatitis, 637.
Hip-joint, Fracture of, Abduction Treatment for, 442; Use of Flexed Plaster Spica Case for, 111.
HITZROT, JAMES M.; Ankylosis of Knee, 609; Post-typhoid Osteomyelitis, 611; Ulcerative Colitis, 612.
Hodgkin's Disease of Bones, 755.
HOMANS, JOHN: Thrombophlebitis of the Lower Extremities, 641.
HOROWITZ, ALEC: Melanotic Tumors, 917.
Horseshoe Kidney, 471.
HORSLEY, J. SHELTON: Some Underlying Principles of Intestinal Surgery, 387.
HUBBARD, WILLIAM B.: Acid and Alkali Burns of the Eye, 89.
Humerus, Intracapsular Fracture of Capitellum of, 789.
HUNT, VERNE C.: Carcinoma of the Ovary in Infancy, 84; Posterior Excision of the Seminal Vesicles, 257.
Hyperadrenalinemia, Adrenalectomy for Alleged, Critique on, 959; in Spontaneous Gangrene, Epinephrectomy (Adrenalectomy) for, 801.
Hypernephroma, Primary, of the Liver, 829.
Hyperthyroidism and Diabetes, 37.
Hypertrophy, Congenital, of the Interureteral Ridge, 751.

I

Ileocæcal Enterocystoma Producing Partial Intussusception, 676.
Ileostomy for Ulcerative Colitis, 799.
Ileum, Terminal, Acute Inflammatory Obstructure of the, 867.
Iliac Vein and Bowel, Bullet Wounds of, 132.
Incisions, Abdominal, 74; Transverse, for Unilateral Hernia, 639.
Injuries, Compound, of Extremities, Treatment of, 321.
Injuries, Heart, with Suture, 823.
Ink-pencil, Indelible, Injuries, 95.
Interureteral Ridge, Congenital Hypertrophy of the, 751.
Intestinal Anastomosis, Aseptic End-to-end, 861.
Intestinal Surgery, Some Underlying Principles of, 387.
Intussusception, Acute, in Infants, 318; Partial, Ileocæcal Enterocystoma, Producing, 676; of Small Intestine into Stomach, 574.
IVY, ROBERT H.: Deformities of the Mandible 596.

J

JAFFE, HENRY L.: Cystic Lymphangioma of the Greater Omentum, 66.
Jaw and Face, Plastic of, Following Excision for Carcinoma, 316.
Jaw, Lower, Fibroma of, 305; Upper, Tumors of the, 306.
JECK, HOWARD S.: Enterolith Suggesting Vesical Calculus, 154.

JENNINGS, JOHN E.: Carcinoma of the Stomach, 951; Suppurative Peri-sigmoiditis, 952; Sympathectomy for Thrombo-angiitis Obliterans, 953.
JOHN, HENRY J.: Hyperthyroidism and Diabetes, 37.
JORDAN, SARA M.: Gastro-jejunal Ulcers and Gastro-jejuno-colic Fistulæ, 231.
JOSEPH, MORRIS: The Prophylaxis of Gas Gangrene in Civil Surgery, 772.

K

Kidney, Horseshoe, 471; Solitary Cysts of the, 250.
Kidney Stones, Operative Removal of, 428.
KING, EDWARD L.: Puerperal Gangrene of the Extremities, 767.
Knee, Ankylosis of, 609.
Knee-joint, Derangements of the, 911.
KULENKAMPFF, PROF. D.: Brachial Plexus Anæsthesia, 883.

L

LAHEY, FRANK H.: Gastro-jejunal Ulcers and Gastro-jejuno-colic Fistulæ, 231; Suture of the Recurrent Laryngeal Nerve, 481.
Laryngeal Nerve, Recurrent, Suture of the, 481.
LASKOWNICKI, STANISLAW: Congenital Hypertrophy of the Interureteral Ridge, 751.
LEE, BURTON J.: Melanoma of Buttock, 621.
Leg, Plastic for Ulcer of, 790.
LEVIN, I. MICHAEL: Abdominal injuries in Children, 718.
LEWISOHN, RICHARD: Acute Perforation of Stomach, 955; Hæmolytic Icterus—Splenectomy, 459; Late Results in Perforated Gastro-duodenal Ulcers, 855; Spontaneous Healing of Osteomyelitis, 456; Thrombocytopenic Purpura—Splenectomy, 458.
LILIENTHAL, HOWARD: Post-pneumonic Lung Abscess Rupturing into Pleura, 624.
Limbs, Blood from Traumatized, and Laked and Traumatized Bloods, 806.
Liver, Non-parasitic Cysts of the, 844; Primary Hypernephroma of the, 829.
Lobectomy for Bronchiectasis, 607.
LOEFFLAD, PAUL A.: Hæmophilia in an Adult, 593.
LOESSL, JOHANN: The De Petz Stomach and Intestinal Suturing Apparatus, 80.
Lung, Infected Congenital Cyst of, 606; Massive Collapse of Right Lung After Nephrectomy, 600; Surgical Treatment of Suppuration of the, 485.
Lung-abscess, Post-pneumonic, Rupturing into Pleura, 624.
Lymphangioma, Cystic, of the Greater Omentum, 66, 872.
Lymph-hæmangiomata, Multiple, 140.
Lymphocytoma, Malignant, 934.
Lymphosarcoma of the mesentery, 879.

M

MACAUSLAND, W. RUSSELL: Poliomyelitis with Especial Reference to Treatment, Edited by. Review of, 477.
Malignant Disease of the Thyroid Gland, 369.
Malignant Lymphocytoma, 934.
Mandible, Deformities of the, 596.
Massive Empyema, Obliterative Operations for, 506.
Maxilla, Superior, Osteofibroma of, 304; Resection of, for Osteosarcoma, 303.
MAYO, CHARLES H.: A New Type of Permanent Colostomy, 711.
MCCREERY, JOHN A.: Fibroma of Lower Jaw, 305; Osteofibroma of Superior Maxilla, 304.
MCGLANNAN, ALEXIUS: Non-parasitic Cysts of the Liver, 844.
Mediastinal Abscess, Posterior, 511.
Melanoma of Buttock, 621.
Melanotic Tumors, 917.
Membranes, Developmental, Causing Obstructive Symptoms, 616.
Meningocele, Cranial, and Spina Bifida, 785.
Mesentery, Fibromata of the, 870; Lymphosarcoma of the, 879.
MILCH, HENRY: Acute Subpectoral Abscess, 517; Indelible Ink-pencil Injuries, 95.
MILLER, M. O.: Puerperal Gangrene of the Extremities, 767.
MILLIKEN, SETH M.: Fracture of Neck of Femur, 787.
MIXTER, W. JASON: Relief of Pain in Carcinoma of the Face, 179.
MONTGOMERY, ALBERT H.: Hodgkin's Disease of Bones, 755.

Moore, George Albert: The Use of Flexed Plaster Spica Case in Hip Fractures, 111.
Morse, Nathan Clark: Exigencies of a General Practice, Review of, 640.
Morton, Charles Bruce: Observations on Peptic Ulcer, 401.
Morton, John J.: Phlegmonous Gastritis, 848.
Muller, George P.: Plastic of Face and Jaw Following Excision for Carcinoma, 316.

N

Narcotics and Anæsthetics, Untoward Effects of, 124.
Neck, Fibro-sarcoma of, 621.
Nelms, Homer L.: Primary Carcinoma of the Hepatic Duct, 157.
Neoplasms and Branchial Anomalies, 186.
Nerve, Recurrent Laryngeal, Suture of the, 481.
Nerves and Tendons, Suture of, in Both Wrists, 309.
Neuhof, Harold: Empyema Following Pneumothorac Therapy for Tuberculosis, 604; Infected Congenital Cyst of Lung, 606; Ileostomy for Ulcerative Colitis, 799; Lobectomy for Bronchiectasis, 607; Recurrent Carcinoma of Chest Wall, 605.
Neuralgia, Trigeminal, Surgical Treatment of, 172.
New York Surgical Society, 140, 292, 296, 455, 604, 619, 787, 950.
Non-malignant Papilloma of the Umbilicus, 636.
Norris, Charles C.: Radium in Gynæcology. Edited by. Review of, 479.

O

Obenour, Sterling W.: Lymphosarcoma of the Mesentery, 879.
Obstruction, Acute Inflammatory, of the Terminal Ileum, 867.
Obstruction of the Bile Ducts, Effects of, 210.
Oersky, M. A.: Brachial Plexus Anæsthesia, 883.
O'Keefe, Charles D.: Sarcoma of Ovary with Unusual Oral Metastases, 467.
Omentum, Cystic Lymphangioma of the Greater, 66; Lymphangioma of, 872.
Operations of Surgery. Edited by R. P. Rowlands and Philip Turner. Review of, 479.
Oppel, Vladimir A.: Epinephrectomy (Adrenalectomy) for Hyperadrenalinemia in Spontaneous Gangrene, 801.
Osteofibroma of Superior Maxilla, 304.
Osteomyelitis, Post-typhoid, 611; Spontaneous Healing of, 456.
Osteoporosis, Traumatic, of the Carpal Bones, 892.
Osteosarcoma, Resection of Superior Maxilla for, 303.
Ovarian Cyst, Rupture of, 307.
Ovary, Carcinoma of the, in Infancy, 84; Sarcoma of, with Unusual Oral Metastases, 467.
Owen, Hubley R.: Carcinoma of the Rectum, 130; Chancre Complicating Laceration of Hand, 783.

P

Pancreas, Accessory, Simulating Gastric Ulcer, 150.
Pancreatitis, Acute Suppurative, 133; Chronic, 637; Chronic, Cholecystenterostomy for, 300.
Papilloma, Non-malignant, of the Umbilicus, 636.
Parsons, Jr., William B.: Polyp of Duodenum, 950.
Patterson, Daniel C.: Transverse Incision for Unilateral Hernia, 639.
Pemberton, John De J.: Embolectomy, 652; Malignant Disease of the Thyroid Gland, 369.
Peptic Ulcer, Observations on, 401.
Perforation, Acute, of Stomach, 955.
Pericholecystitis, Anomalous Hepatic Duct, 151.
Perinephritic Abscess, 301.
Peri-sigmoiditis, Suppurative, 952.
Peritoneal Cavity, Lesser, Hernia into, 312.
Peterson, Edward W.: Cholelithiasis and Large Umbilical Hernia, 299; Congenital Goitre, 297; Graves' Disease in the Male, 296.
Pfeiffer, Damon: Rupture of Ovarian Cyst, 307.
Phelps, Alfred E.: Coronary Disease in Surgical Patient, 32.
Phemister, Dallas B.: Traumatized and Laked Bloods and Blood from Traumatized Limbs, 806.
Philadelphia Academy of Surgery, 130, 307, 593, 779.
Phlegmon, Sublingual, 684.

Phlegmonous Gastritis, 848.
PICKFORD, EDWARD M.: Intussusception of Small Intestine into Stomach, 574.
PICKHARDT, OTTO C.: Concomitant Gastric and Duodenal Ulcers Two and one-half Years Post-operative, 143; Lung Abscess Following Pneumococcus Septicæmia, 141; Multiple Lymph-hæmangiomata, 140.
Pneumectomy, Cautery, 159.
Pneumococcus Septicæmia, Lung Abscess Following, 141.
Poliomyelitis with Especial Reference to Treatment. Edited by W. RUSSELL MACAUSLAND. Review of, 477.
Polya-Mayo Exclusion for Duodenal Ulcer, 301.
Polyp of Duodenum, 950.
Popliteal Aneurism, Ruptured, 455.
Post-typhoid Osteomyelitis, 611.
Pott's Fracture, Suspension of the Foot in, 450.
Pregnancy, Extra-uterine, Surgical Intervention in, 581.
Prostatic Middle Lobe Hypertrophy, 785.
Pseudo-carcinoma of the Stomach, 555.
Puerperal Gangrene of the Extremities, 767.
Pulmonary Embolism, 528.
Punctures, Ventricular, 1.
Pyloroplasty for Perforated Duodenal Ulcer, 619.

Q

QUAIN, ERIC P.: Prophylactic Gastrostomy, 395.

R

Radium in Gynæcology. Edited by JOHN G. CLARK and CHARLES C. NORRIS, and GIOACCHINO FAILLA. Review of, 479.
Radius, Formation of, Congenitally Absent, 105.
RAINE, FORRESTER: Untoward Effects of Narcotics and Anæsthetics, 124.
RANDALL, ALEXANDER: Prostatic Middle Lobe Hypertrophy, 785.
RATHBUN, NATHANIEL P.: Horseshoe Kidney, 471.
Rectum, Carcinoma of the, 130, 790.
Recurrent Laryngeal Nerve, Suture of the, 481.
REDER, FRANCIS: Non-malignant Papilloma of the Umbilicus, 636.
ROEDER, CLYDE A.: Acute Inflammatory Obstruction of the Terminal Ileum, 867.
ROGOFF, J. M.: Critique on Adrenalectomy for Alleged Hyperadrenalinemia, 959.
ROLLINGS, JR., H. W.: Diverticula of the Urinary Bladder, 742.
ROWLANDS, R. P.: Operations of Surgery, Edited by. Review of, 479.
Rupture, Traumatic, of Urinary Bladder in Children, 307.

S

SANTEE, HAROLD E.: Ulcerative Colitis, 704.
Sarcoma of Ovary with Unusual Oral Metastases, 467; Primary, of the Clavicle, 382.
Scaphoid, Tarsal, French-heel Fractures of the, 587.
SCOTT, ERNEST: Lymphosarcoma of the Mesentery, 879.
Seminal Vesicles, Posterior Excision of the, 257.
SEMKEN, GEORGE H.: Tumors of the Upper Jaw, 306.
SCHOENFELD, HERBERT H.: Heart Injuries with Suture, 823.
SHALLOW, THOMAS A.: Spina Bifida and Cranial Meningocele, 811; Spina Bifida and Cranial Meningocele, 785.
SHARPE, WILLIAM: Ventricular Punctures, 1.
SHEARER, JOHN P.: Intussusception of Small Intestine into Stomach, 574.
SHEN, JAMES K.: Aseptic End-to-end Intestinal Anastomosis, 861.
SHIPLEY, ARTHUR MARRIOTT: Progressive Gangrenous Ulceration of the Abdominal Wall, 245.
SHORE, BENJAMIN RICE: Gastro-jejunocolic Fistula, 578.
Sigmoiditis, Acute, 783.
SILVER, PHILIP G.: Malignant Lymphocytoma, 934.
SIMON, HAROLD E.: Carcinoma of the Ovary in Infancy, 84.
Skin, Recurrent Fibromatous Tumors of the, 945.
Small Intestine, Intussusception of, into Stomach, 574.
SMITH, MORRIS K.: Strangulated Umbilical Hernia with Gangrene of Transverse Colon, 144.
SMYTH, JR., CALVIN M.: Hernia into Lesser Peritoneal Cavity, 312; Reduction "En Bloc" of Strangulated Hernia, 313.

Spina Bifida and Cranial Meningocele, 785, 811.
Splenectomy for Blood Dyscrasia, 130; Hæmolytic Icterus, 459; for Pernicious Anæmia, 314; Thrombocytopenic Purpura, 458.
Spongioblastoma Multiforme of the Brain, 8.
STABINS, SAMUEL J.: Phlegmonous Gastritis, 848.
STETTEN, DEWITT: Intraperitoneal Rupture of Urinary Bladder During Abdominal Examination, 465; Multiple Complications Following Appendicectomy, 461; Resection and Reconstructive Arteriorrhaphy, 462.
STILLMAN, ALFRED: Perforating Duodenal Ulcer, 292; Ruptured Popliteal Aneurism, 455.
STIRLING, CALHOUN: Diverticula of the Urinary Bladder, 742.
Stomach, Acute Perforation of, 955; Carcinoma of the, 951; and Duodenum, Ulcer of the, Treatment of, 566.
Stomach and Intestinal Suturing, Apparatus, the De Petz for, 80; Pseudo-carcinoma of the, 555.
Stone, Kidney, Operative Removal of, 428.
STOOKEY, BYRON: Surgical Treatment of Trigeminal Neuralgia, 172.
STOUT, ARTHUR PURDY: Branchial Anomalies and Neoplasms, 186.
Sublingual Phlegmon, 684.
Subpectoral Abscess, Acute, 517.
Suppuration of the Lung, Surgical Treatment of, 485.
Surgery, Biliary, Remote Results of, 837; Civil, the Prophylaxis of Gas Gangrene in, 772.
Surgical Patients, Coronary Disease in, 32.
Sympathectomy for Thrombo-angutis Obliterans, 953.

T

Tannic Acid Treatment of Burns, 292.
Tarsal Scaphoid, French-heel Fractures of the, 587.
TAYLOR, ALFRED S.: Developmental Membranes Causing Obstructive Symptoms, 616.
TAYLOR, KEMPTON P. A.: Undescended testicle, 594.
Tendons and Nerves, Suture of, in Both Wrists, 309.
Testicle, Malignant Tumors of the, 263; Undescended, 594.
THEIS, FRANK V.: Ileocæcal Enterocystoma Producing Partial Intussusception, 676.
Thrombo-angiitis Obliterans, Sympathectomy for, 953.
Thrombocytopenic Purpura—Splenectomy, 458.
Thrombophlebitis of the Lower Extremities, 641.
Thyroid Gland, Malignant Disease of the, 369.
Tonsillectomy with Bloodless Technic, 378.
Transfusion, Blood, the Technic of, 775.
Trigeminal Neuralgia, Surgical Treatment of, 172.
TROUTT, JAMES M.: The Technic of Blood Transfusion, 775.
Tumors, Brain, Removal of, 779; Malignant, of the Testicle, 263; Melanotic, 917; Recurrent Fibromatous, of the Skin, 945; of the Upper Jaw, 306.
TURNER, PHILIP: Operations of Surgery. Edited by. Review of, 479.
Typhoid Perforation, Enterostomy in, 476.

U

Ulcer, Duodenal, Polya-Mayo Exclusion for Duodenal, 301; of leg, Plastic for, 790; Observations on Peptic, 401; Perforating Duodenal, 292; Perforated Duodenal, Pyloroplasty for, 619; of the Stomach and Duodenum, Treatment of, 566.
Ulceration, Progressive, Gangrenous, of Abdominal Wall, 245.
Ulcerative Colitis, 612.
Ulcers, Gastric and Duodenal, Concomitant, Two and One-half Years Post-operative, 143.
Ulcers, Gastro-jejunal, and Gastro-jejunocolic Fistulæ, 231.
Ulcers, Late Results in Perforated Gastroduodenal, 855.
Umbilical Hernia, Large and Cholelithiasis, 299.
Umbilicus, Non-malignant Papilloma of the, 636.
Undescended Testicle, 594.
Urinary Bladder, Intraperitoneal Rupture of, During Abdominal Examination, 465.

Urology and Syphilology, Practice of, Edited by CHARLES H. CHETWOOD. Review of, 478.

V

VAN WAGENEN, WILLIAM P.: Sublingual Phlegmon, 684.
VANDER VEER, EDGAR A.: Primary Carcinoma of the Hepatic Duct, 157.
Ventricular Punctures, 1
Vesical Calculus, Enterolith Suggesting, 154.
Vesicles, Seminal, Posterior Excision of the, 257.
Visor Flaps from the Chest, 364.

W

WAGNER, LEWIS CLARK: Posterior Mediastinal Abscess, 511.
WALKER, W. WALLACE: Tonsillectomy with Bloodless Technic, 378.
WANGENSTEEN, OWEN H.: Cholangitis Following Cholecystenterostomy, 54.
WEEDER, DANA: Suture of Tendons and Nerves in Both Wrists, 309.
WEHRBEIN, H. L.: Cystoscopy Reactions, 435.
WHITE, WILLIAM CRAWFORD: Pyloroplasty for Perforated Duodenal Ulcer, 619.
WHITEFORD, C. HAMILTON: Cautery-pneumectomy, 159.
WHITMAN, ROYAL: Abduction Treatment for Fracture of Neck of Femur, 320, 442.
WHIPPLE, ALLEN O.: Carcinoma of the Rectum, 790.
WILLIS, BYRD C.: Congenital Dilatation of Common Bile Duct, 48.
WILLIS, DAVID A.: Recurrent Fibromatous Tumors of the Skin, 945.
WRIGHT, V. W. MURRAY: French-heel Fractures of the Tarsal Scaphoid, 587.
Wrists, Suture of Tendons and Nerves in Both, 309.

Y

YATER, WALLACE M.: Acquired Arteriovenous Fistula, 19.
YATES, JOHN LAURENCE: Untoward Effects of Narcotics and Anæsthetics, 124.

GASTRON

Steadily advances in use and repute

As it becomes more and more widely known

GASTRON fortifies gastric digestion, relieving and correcting disorder of gastric function. It is also more and more employed as an accessory to other treatment—to enable the patient to get the maximum of nutrition, and to promote tolerance of remedies.

GASTRON—the acid-aqueous-glycerin extract of the entire gastric mucous membrane*—prescribed simply by the name GASTRON.

Samples, literature, upon request.

Fairchild Bros. & Foster

*Alcohol free. **New York**

Valentine's Meat-Juice

For **Quieting** the **Irritable Stomach** in **Pregnancy,** for **Rapidly Restoring** the **Vital Forces** in **Hemorrhage,** for **Sustaining** and **Strengthening** in **Long** and **Exhausting Labor, Valentine's Meat-Juice** is extensively employed in

Obstetrical Practice.

Dr. Karl A. Schetelich, *Physician to the City Orphanage, Chemnitz, Germany:* "I have employed VALENTINE'S MEAT-JUICE with the best results with women in childbirth after severe hemorrhages."

Prof. Dr. A. Jentzer, *Professor of Gynaecological Clinic, University of Geneva, Switzerland:* "I have used VALENTINE'S MEAT-JUICE in the treatment of Women weakened by Puerperal Hemorrhages. The preparation was well retained, well assimilated and the patients regained their strength very rapidly."

For Sale by American and European Chemists and Druggists.

VALENTINE'S MEAT-JUICE CO.,

411-K **RICHMOND, VIRGINIA, U. S. A.**

Why The Phenolphthalein?

In colon stasis the action of mineral oil is *merely* mechanical. It exerts no therapeutic action.

Agar-agar absorbs moisture and increases fecal bulk, thus intensifying peristaltic reflexes—but it does not exert direct action.

Phenolphthalein is a positive peristaltic stimulant, which also increases the secretion of the intestine without producing pain, and it has no action on any other organs.

The soundness of including it in the original mineral oil and agar-agar emulsion has been conclusively proved by the striking success and increasing popularity with the profession of

AGAROL is the *original* Mineral Oil—Agar-Agar Emulsion and has these special advantages:

Perfectly homogenized and stable; pleasant taste without artificial flavoring; freedom from sugar, alkalies and alcohol; no contraindications; no oil leakage; no griping or pain; no nausea or gastric disturbances; not habit forming.

AGAROL

A Generous Trial Quantity Free Upon Request

WILLIAM R. WARNER & CO., INC.

Manufacturing Pharmaceutists since 1856

113-123 West 18th Street -:- New York City

HOW WILL THE MORNING FIND YOUR PATIENT?

THE selection of the right hypnotic is no longer a problem with thousands of physicians who know the many advantages of

DIAL, "CIBA"

(Diallylmalonylurea)

Hypnosis without after effects—Safety with effectiveness.

TABLETS AMPULES LIQUID

Descriptive Literature and Specimen upon Request.

CIBA

PURITY RELIABILITY

CIBA COMPANY

Incorporated

Cedar and Washington Streets New York City

Canada: 146 St. Peter Street, Montreal